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A CLINICAL STUDY OF SILICOSIS

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THIS is a report of a clinical investigation of 203 miners working in a large gold mine in Ontario. The investigation was undertaken in the hope that the clinical findings might advance the knowledge of the diagnosis, prognosis and therapy of silicosis, and in this way aid in the prevention of the condition. At the time of examination all the miners were working and none were known to be tuberculous.

For a considerable time it has been apparent to those who are familiar with the problem of silicosis that markedly different reactions exist amongst those who are constantly breathing a dust containing silica. These differences are especially impressive when one frequently notices that two miners of approximately the same age, the same underground experience, with equal dust exposure, with like habits, good and bad, with like living conditions, diet, etc., are unlike in one respect, that is, that one man develops silicosis while the other man goes on for an equal time or years longer with very little abnormal change in his chest.

While much has been written on the subject of silicosis little has been done in the field of clinical study of the individual silicotic. It was with this thought in mind that early in 1934 the problem of investigating a group of miners was undertaken. This group embraced the ratings of the Workmen's Compensation Board of Ontario in the classification of chests, namely,—more fibrosis (MF), more fibrosis plus (MFP), more fibrosis plus plus (MFPP), and silicosis.

The data collected on each individual included age, particular occupation, time of exposure to dust, a complete history and physical examination. Red, white, differential and Arneth polymorphonuclear counts, hæmoglobin estimation, and sedimentation rate of the erythrocytes were done routinely. Further blood analysis included non-protein nitrogen, urea, uric acid, sugar, calcium, phosphorus, cholesterol, soluble silica, Wassermann tests, van den Bergh or icterus index, CO_2 combining power, and blood culture. These estimations were all made on the blood of fasting patients and done immediately in our own laboratory. In addition the examination included complete urine and sputum analysis and cultures from the nose, throat, gums, urine and sputum. The basal metabolic rate and electrocardiagrams were done as a routine. X-ray films of the chest were made on all cases.

The men examined were divided into the following groups on the basis of the Workmen's Compensation Board classification.

TABLE I.

Group	*Rating*	*Number men*	*Average age*	*Average years of dust exposure*
1	Silicosis	48	46.2	16.9
2	MFPP	31	44.0	19.7
3	MFP	41	37.3	8.62
4	MF	36	31.0	5.27
5	MF and MFP	23	45.0	13.4
6	MF and MFP	24	37.0	5.09

Examination of the nervous system showed nothing abnormal. No symptoms referable to thyroid dysfunction were present in any case. No man examined gave a history of contact with tuberculosis. Routine urinalysis did not show anything of significance. Sputum smears

TABLE II.

PERCENTAGE OF NATIONALITIES

Group	British Canadian	American	Austrian Czecho-Slovakian Jugo-Slav	Belgian	Bulgarian	Finlander	Italian	Lithuanian	Polish	Roumanian	Russian Ukrainian	Spanish	Others
1	45.83	2.08	10.45	0	4.16	0	12.50	2.08	2.08	4.16	14.58	2.08	0
2	54.84	0	12.90	0	6.45	3.23	6.45	0	3.23	6.45	6.45	0	0
3	41.46	0	39.03	0	0	0	17.07	0	0	2.44	0	0	0
4	50.00	0	38.89	0	0	2.78	5.55	0	0	2.78	0	0	0
5	47.82	0	26.09	0	0	0	21.74	0	0	4.35	0	0	0
6	62.50	0	29.17	4.16	0	0	0	0	4.17	0	0	0	0
Total now employed 2,041	64.97	0.39	19.56	0.10	0.10	4.64	4.69	0.44	1.67	1.08	0.69	0.05	1.62

TABLE III.

RESPIRATORY SYSTEM

	Dyspnœa		Colds			Chest pain			Cough			Sputum			
Group	Positive	Average time years	Frequent	Occasional	None	Frequent	Occasional	None	Frequent	Occasional	None	Moderate	Occasional	None	Blood stained
	%	%	%	%	%	%	%	%	%	%	%	%	%	%	%
1	87.2	3.60	21.3	53.2	25.5	32.0	55.3	25.5	50.0	15.2	34.8	41.3	13.0	45.7	15.2
2	48.3	2.96	31.0	31.0	38.0	6.9	17.2	75.9	24.1	17.2	58.7	17.2	6.9	75.9	3.4
3	14.6	1.60	14.6	53.7	31.7	0	4.9	95.1	12.2	19.5	68.3	9.8	2.4	87.8	0
4	11.1	2.17	11.1	52.8	36.1	0	2.8	97.2	2.8	2.8	94.4	0	2.8	97.2	0
5	26.1	1.75	26.1	43.5	30.4	0	4.4	95.6	17.4	17.4	65.2	8.7	2.8	91.3	0
6	33.3	3.36	16.7	58.3	25.0	16.7	0	83.3	16.7	29.2	54.1	16.7	12.5	70.8	4.1

TABLE IV.

	Gastro-intestinal	Urinary night frequency		Liquor			Tobacco				Diet				
Group	Functional upsets	Occasional	Once or twice nightly	None	Occasional	Moderate	None	Pipe	Cigarettes	No. Cigarettes daily	Carbohydrate		Protein		Balanced
											% High	% Low	% High	% Low	
	%	%	%	%	%	%	%	%	%						%
1	31.25	17.4	4.3	21.4	36.0	42.6	17.0	17.0	66.0	12	12.8	0	2.1	12.8	72.3
2	16.1	6.9	3.4	17.2	65.6	17.2	24.1	13.8	62.1	17	13.8	0	3.4	20.7	62.1
3	19.5	2.4	0	14.6	51.2	34.2	7.3	7.3	83.0	15	2.4	2.4	4.8	0	90.4
4	8.3	2.8	0	16.7	58.3	25.0	16.7	8.3	75.0	14	0	0	5.6	2.8	91.6
5	30.4	8.7	4.3	17.4	39.1	43.5	13.0	17.4	65.2	13	0	0	4.4	0	95.6
6	4.17	4.2	0	25.0	41.7	33.3	12.5	12.5	66.7	15	0	0	12.5	12.5	75.0

TABLE V.

	Teeth				Gums		Tonsils		
Group	Good	Fair	Bad	Artificial	Pyorrhœa	Healthy	Out	Not apparently diseased	Apparently diseased
	%	%	%	%	%	%	%	%	%
1	17.0	25.5	40.5	17.0	72.3	27.7	6.4	85.1	8.5
2	20.7	31.0	31.0	17.3	65.5	34.5	24.1	71.4	4.5
3	31.7	31.7	34.2	2.4	65.8	34.2	7.3	92.7	0
4	44.4	33.3	22.3	0.0	58.3	41.7	5.6	94.4	0
5	8.7	34.8	52.2	4.3	87.0	13.0	4.4	95.6	0
6	37.5	41.7	12.5	8.3	54.2	45.8	12.5	87.5	0

TABLE VI.

THROAT, NOSE, GUMS: CULTURES

	Throat				*Nose*				*Gums*			
Group	*NHS*	*HSA*	*SV*	*Etc.*	*HSA*	*NHS*	*SV*	*Etc.*	*SV*	*NHS*	*HSA*	*Etc.*
1	84.8	0	10.8	4.4	89.1	0	0	4.4	71.7	23.9	0	4.4
2	86.0	0	3.5	10.5	79.3	0	3.5	17.2	79.2	6.9	6.9	7.0
3	87.8	0	9.7	2.5	79.5	0	0	20.5	84.6	13.0	0	2.4
4	75.0	3.6	10.7	10.7	85.7	3.6	0	10.7	81.5	14.8	0	3.7
5	86.4	0	9.1	4.5	80.9	0	0	19.1	90.4	4.8	0	4.8
6	87.5	0	8.3	4.2	66.7	4.2	0	29.1	70.8	25.0	4.2	0

Predominant Organisms:
NHS—Non-hæmolytic streptococcus
HSA—Hæmolytic staphylococcus aureus
SV—Streptococcus viridans
Etc.—Includes *B. Coli*, micrococcus catarrhalis, hæmolytic streptococcus, staphylococcus albus.

TABLE VII.

	Cytology			*Smears*			*Arneth polymorphonuclear*			
Group	*Hb.*	*Average R.B.C.*	*Average W.B.C.*	*Poly.*	*Lymph.*	*Eosin.*	*I*	*II*	*III*	*IV*
	%			%	%	%				
1	85.7	4,065,000	7005	54	45	1	34	44	20	2
2	88.0	4,289,000	7066	60	39	1	34	41	22	3
3	91.4	4,401,000	6780	56	43	1	32	45	20	3
4	92.5	4,408,000	7506	59	40	1	35	44	19	2
5	93.1	4,470,000	6501	60	39	1	33	47	18	2
6	89.7	4,345,000	6809	54	44	2	30	42	23	5

TABLE VIII.

		Blood pressure		
Group	*Pulse*	*Average diastolic*	*Average systolic*	*Basal metabolism rate*
1	58.7	84.6	124	minus 7.06
2	58.4	85.2	125	minus 7.9
3	69.8	83.7	124	plus 9.6
4	73.6	83.75	127	plus 11.5
5	69.9	81.7	122	plus 9.4
6	62.6	84.8	127.6	minus 7.42

TABLE IX.

BLOOD CHEMISTRY

Group	*N.P.N.*	*Urea nitrogen*	*Uric acid*	*Sugar*	*Icterus index*	*van den Bergh*	*CO_2 Combining power (plasma)*	*Inorganic calcium*	*Inorganic phosphorus*	*Cholesterol*	*Soluble silica*	*Wassermann positive*	*Sedimentation 1 hour*
												%	
1	34.77	16.0	2.94	109.7	6.7	0.37	55.95	10.77	3.47	158.4	1.04	2.08	24.3
2	33.2	14.88	2.81	109.6	6.2	0.25	59.0	10.73	3.29	160.8	0.85	3.23	15.3
3	37.35	16.86	2.73	110.75	7.0	0.30	55.0	10.77	3.53	160.1	1.03	0	11.3
4	32.2	14.37	3.12	111.47	6.6	0.32	55.2	10.60	4.14	156.3	0.93	11.1	8.3
5	37.2	16.5	2.65	110.8	7.9	0.25	56.5	10.97	4.10	160.6	1.02	0	17.3
6	37.0	16.78	2.73	111.08	5.7	0.32	53.9	10.29	4.05	167.3	0.94	13.0	10.7

and cultures were all negative for tubercle bacilli at the time of examination.

Heart.—A group of 50 silicotics and an equal number of non-silicotics were examined. The results are as follows:

TABLE X.

	Silicotics	*Non-silicotics*
No. examined	50	50
Average age	47.5	45.8
Average years mining	18	17.3
Dyspnœa	39	21
Evidence of heart disease clinically	7	10
Hearts	*Percentage*	*Percentage*
Vertical	51.0	57.7
Hypoplastic	14.3	3.8
Horizontal	26.5	21.2
Globular	8.2	17.3

Electrocardiagrams.—

TABLE XI.

	Silicotics	*Non-silicotics*
Definite evidence of heart disease	6	3
Suggestive evidence of heart disease	7	4
L.A.D.	15	21
R.A.D.	2	1
25 absent or short	40	44
Positive T5	9	10
High T1 or 2	16	15
Prolonged conduction	1	0
Inverted T1 or 2	1	2

Heart measurements.—

TABLE XII.

	Silicotics		*Non-silicotics*	
	Transverse diameter		*Transverse diameter*	
Year	*Actual*	*Normal*	*Actual*	*Normal*
1931	132.5 mm.	124.1 mm.	137.7 mm.	127.3 mm.
1936	134.1 mm.	125.1 mm.	139.7 mm.	129.8 mm.

The above measurements were made on 33 silicotics and 44 non-silicotics from x-ray films taken at a distance of 52 inches. The normal diameters were estimated from a table based on a film distance of 72 inches.

In view of the consistently low basal metabolic rates and the high blood sedimentation rates in the silicotic group, 30 of these men were given thyroxine tablets, 0.2 mg. daily for one month, then increased to 0.4 mg. daily for eight to ten months. At the end of that time a re-check was made of the basal metabolic rates, the blood sedimentation rate, calcium, phosphorus and cholesterol.

TABLE XIII.

	Before	*After*	
Basal metabolism	minus 7.06	gain 58.1%	plus 9.8
		loss 35.5%	minus 6.8
		same 3.3%	
Blood sedimentation	24.3 mm.	drop 72.4%	minus 14.2 mm.
		up 24.1%	
		same 3.5%	
Calcium	10.77	11.17	
Phosphorus	3.47	3.32	
Cholesterol	158.4	177.3	

The basal metabolic rate increased 9.8 per cent in 58.1 per cent and decreased on an average of 6.8 per cent in 35.5 per cent, while 3.3 per cent remained the same.

The blood sedimentation rate is interesting, in that from the average figure before thyroid medication of 24.3 mm. in one hour, 72.4 per cent dropped an average of 14.2 mm.; 3.5 per cent remained the same; while 24.1 per cent, or 7, increased. Of these 7, four increased less than 5 mm. and 1 increased 6 mm. Of the remaining 2, one increased from 21 to 70 and the other from 46 to 94. Examination revealed that both these men had developed pulmonary tuberculosis. They were removed from work and recommended for sanatorium treatment. It is an interesting observation that in some cases where the thyroid medication did not raise the basal metabolic rate and where it actually dropped lower the blood sedimentation rate dropped with clinical improvement.

After this group of 30 men had received thyroid therapy for eight to ten months, their chests were x-rayed and the films compared with films taken when the thyroid was started. The chest plates of two men showed evidence of chronic infection not previously seen. In one case a positive sputum was obtained where previous examinations of the sputum showed no tubercle bacilli.

While no evidence of clinical improvement was seen in the chest picture, except in one case, there was no apparent advance of the fibrosis. The interpretation of these films was made by Dr. N. H. Russell, for whose cooperation we are much indebted.

DISCUSSION

In Table I is a classification of the nationalities of the men examined and also of the men now employed underground at this mine.

Those of British extraction predominate in all groups and are about equal. There is a high percentage of Italians and Russians in group 1. The percentage of Italians in groups 3 and 5 is higher, while there are no Russians in groups 3, 4 or 5. The percentage of those of the Austrian group is lower in group 1 than in other groups. The numbers in other groups is so small as to be of little significance.

The symptoms referable to the respiratory system are seen in Table II. Under the heading of Dyspnœa 87.2 per cent of the 1st group gave a positive history, with a decrease in the incidence to 11.1 per cent in group 4. Group 5 showed 26.1 per cent of positives. As dyspnœa is one of the first and most important symptoms encountered in silicosis these figures are about average. Under the heading of "colds", embracing respiratory infections generally, the figures apparently do not carry any particular significance. Considering chest pain, the columns headed "frequent" and "occasional" indicate the high incidence of chest pain in group 1, as the total of these two columns in this group is equal to 87.3 per cent, with groups 2 and 6 next in frequency. Likewise in the study of cough as a symptom the figures are almost as striking as in the study of chest pain, as group 1 shows 65.2 per cent having coughs as compared with the next highest figure of 45.9 per cent in group 6 and 41.3 per cent in group 2. The last respiratory feature considered was sputum. Again the difference between group 1 and the other groups is very marked; in that group 1 showed 54.3 per cent having sputum, as compared with 24.1 per cent of group 2 and 29.2 per cent in group 6. Blood-stained sputum was found in 15.2 per cent of group 1, 3.4 per cent of the 2nd group, 4.1 per cent in group 6, and not at all in any of the other groups. Thus it is obvious that the symptoms referable to the respiratory system are higher in groups 1, 2 and 6.

The diet, the quantity of liquor and tobacco consumed apparently were not etiological factors in any group.

The condition of the teeth and gums in all the groups examined was very poor. Less than 45 per cent of teeth could be classified as good, with group 1 showing only 17 per cent good, and group 5 falling to the level of 8.7 per cent. The presence and severity of the pyorrhœa was likewise marked, in that the best group, namely 6, showed 54.2 per cent pyorrhœa, increasing to 72.3 per cent among group 1, and with group 5 heading the list with 87 per cent pyorrhœa. There is obviously a marked need for dental attention and supervision amongst our mining group.

The results of bacterial culture from nose, throat and gums show comparatively little variation from group to group, but it is interesting to note that the predominating organism in the throat in 84.8 per cent of cases was a non-hæmolytic streptococcus; the predominating organism in 89.1 per cent of noses was a hæmolytic *Staph. aureus*; and, lastly, the predominating organism from 71.7 per cent of gums was a *S. viridans*.

In Table VII it is seen that there is a drop from 93.1 per cent hæmoglobin in group 5 to 85.7 per cent in group 1. Also, there is a drop from 4,470,000 to 4,065,000 in the red blood count. This would seem to indicate a slight degree of anæmia peculiar to group 1. The white blood count shows such variation as to suggest that it is not significant. The blood smears and Arneth polymorphonuclear counts show little variation from group to group.

A study of the pulse rates, taken while the various men were resting for basal metabolism estimation, is of interest. In group 1 the average pulse while resting was 58.7; in group 2 it was 58.4; in group 3 it was 69.8; in group 4 it was 73.6; in group 5 it was 69.9; and in group 6 the rate was 62.6. The consistently low pulse rate in those of group 1 and 2 seemed to distinguish them very definitely from those of group 3 and 4.

A study of average systolic blood pressures shows such readings to be very close to one another.

The basal metabolic rates gave us one of the most interesting observations on this group of miners, and it was upon these findings that some treatment suggested itself. Groups 1, 2 and 6 showed lowered basal rates, which averaged –7.06, –7.9 and –7.42, respectively, whereas the 3rd, 4th, and 5th groups gave readings averaging plus 9.0, plus 11.5, and plus 9.4, respectively.

The blood non-protein nitrogen, urea nitrogen, uric acid, calcium, phosphorus and cholesterol showed very little variation in any of

the groups. Apparently the kidney is not affected by the presence of silica in the body, as both the 3rd and 5th groups gave higher average non-protein nitrogen and blood urea figures than did group 1. There is very little variation in the blood sugar readings. With reference to the liver it would seem that the similarity in value for icterus index and van den Bergh, well within normal limits, leads one to conclude that liver damage from silica in the body is negligible. There is very little variation in the CO_2 combining-power values in all the groups, and the figures for blood silica vary so much as to be apparently of little significance.

There is apparently no relationship effect of syphilitic infection on any of these groups. Group 1 showed the highest average blood sedimentation rate, namely, 24.3, with group 5 next with 17.3 mm. The other groups fall from 15.3 mm. for group 2, to 8.3 mm. in the 4th group. The men in group 5 were selected from groups 3 and 4 for comparison with group 1, as they were of a similar age and dust-exposure time.

The men in group 6 are classified as MF and MFP. The average age of these two groups was 37 years. Although the average underground experience of the MFP group was 11.27 years, a close analysis of the occupations in which there is dust exposure of any serious degree showed that during their underground experience their dust contact averaged only 5.09 years. A comparable figure for the MF group shows an underground experience of 9.83 years with average exposure to dust in serious quantity of only 4.38 years. The other groups considered in this paper showing plus basal metabolic rates are made up of machine men, runners, blasters, and loaders, while these latter two groups with negative basal rates are for the most part trackmen, cage-tenders, diamond drill workers, etc., with comparatively little dust contact. We feel that this small dust exposure may be of considerable significance in view of their negative basal rates, and it is felt that with increased dust exposure they might have shown progress in fibrosis, whereas our 3rd and 4th groups and group 5 with positive basal rates and much dust exposure have not progressed as group 1 and group 2 have.

SUMMARY

Apparently silicotics and MFPP differ from those not affected by advanced fibrosis in that they showed a lowered basal metabolic and pulse rate.

The blood sedimentation rate was consistently higher in silicotics than in other groups.

There was a consistent decrease in the blood sedimentation rate in a large proportion of silicotics after the administration of thyroxine.

The condition of the teeth in all groups showed the need of oral therapy.

CONCLUSIONS

It may be possible to recognize the miner who will develop silicosis, before the appearance of the usual signs, by lowered basal metabolism and pulse rates.

Thyroid therapy may possibly not only prevent the development of silicosis, but may also arrest its further progress, and possibly reduce to some extent the degree of fibrosis already existing.

Diet, social and living conditions amongst miners are quite uniform, and evidently do not affect the development of silicosis.

In the study of these miners there is no evidence to show that liver, kidney or heart disease was associated with dust inhalation.

A large proportion of silicotics have shortness of breath in varying degrees. All the silicotics studied had a lowered basal metabolic rate and slow pulse, which might be a compensating factor in reducing cardiac output with lessened heart strain, and account for the large number of normal-shaped hearts in cases of long-standing silicosis, contrary to what one would expect in the presence of diffuse lung fibrosis.

The presence of definite evidence of heart disease in a silicotic disabled from shortness of breath would certainly point to the fact that part of the disability is due to heart disease but we know of no way of determining what proportion of the disability should be apportioned to one or other cause of the disability.

This study has revealed valuable clinical information, and is being continued with the hope that future work may aid in the solution of the complex problem of silicosis.

This work was done at the Hollinger Consolidated Gold Mines, Ltd., Timmins, Ontario.

We appreciate the assistance of the Department of Health Laboratory, Toronto, of Dr. N. H. Russell, who interpreted the x-ray films, and of Dr. John Hepburn for interpretation of the electrocardiagrams.

GASTROSCOPY: ITS INDICATIONS AND VALUE*

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ACCURATE scientific diagnosis of visceral lesions has been made possible only within the past fifty years. Early lesions which in other days were completely neglected are now seen daily by direct inspection through endoscopic instruments. While indirect methods of radiographic contrast are of the utmost importance no one will deny that direct visual inspection, when possible, is the method of choice. It is only by the use of the endoscopic method that one can hope to diagnose very early such lesions as carcinoma of the bladder, rectum, bronchus or stomach.

In the study of diseases of the stomach a thorough history and an expert radiological examination are, of course, of prime importance. Further progress in the earlier diagnosis of inflammatory lesions or malignancy must depend upon direct visualization. Gastroscopy is almost indispensable in the diagnosis of gastritis, although valuable information may be obtained from roentgenographic mucosal relief. X-ray evidence of gastric ulcer will sometimes disappear long before the complete resolution of the lesion is observed through the gastroscope. Further, in 121 consecutive gastroscopies we were able to demonstrate definite gastric ulcers in 5 patients in whom the x-ray examination was negative. It is in the study of gastric carcinoma that we hope for most striking results. Of the 3,000 people who die of carcinoma of the stomach in Canada each year only a few are operable when seen by the surgeon, and in the most expert hands less than 10 per cent are cured. Surely direct visualization of the gastric mucosa will offer us a better chance of early and accurate diagnosis. The removal of a specimen of diseased tissue for biopsy is not commonly practicable through the gastroscope at present. In the future it will become more practicable, and endoscopy and biopsy will undoubtedly lead to life-saving accuracy and early diagnosis of gastric lesions. The more we develop the technique of endoscopy and biopsy in diseases of the hollow viscera, the more definite and early will become our diagnosis, and the more direct and accurate our surgical attack.

THE HISTORY OF GASTROSCOPY

It was in 1865 that Desormeaux wrote "One of the characteristic advancements of our time lies in the search for methods to explore the viscera directly, in the careful study of local lesions by the aid of these methods, and in the practical application of knowledge thus gained to the medical sciences. We ask science to furnish us with instruments of precision which will serve to explore and give us greater knowledge of the organs". Desormeaux was a pioneer in urethroscopy, cystoscopy, and proctoscopy. Since his original contributions, over seventy years ago, the extension of this method of medical investigation has revolutionized diagnostic procedures. In 1868 Kussmaul first attempted gastroscopy upon a professional sword-swallower. The first gastroscopy that was partly successful was done by Trouve in 1871. In 1881 Mikulicz became interested in this method of examination, and in 1883 was able to describe the appearance of malignant invasion of the pyloric antrum of the stomach. Nitze and Littré constructed a flexible gastroscope in 1879-1881, and in the following years improvements in such instruments were made by Rosenheim, Killing and Kutner. The next definite improvement was made by Rewidzoff, who attached a flexible segment as a guide to the rigid type of tube. During the next twenty years many investigators made noteworthy contributions to the perfecting of the gastroscope, both in regard to safety and visibility. Special mention must be

* Presented at the Meeting of the Royal College of Physicians and Surgeons of Canada, at Ottawa, October 31, 1936.

made of the work of Elsner and Martin Sussman, who not only improved the mechanical construction of the instrument but further developed its clinical application. Since 1920 we are especially indebted to the ingenuity and persistence of Schindler, Korbsh, Henning, C. Jackson, Moutier and others.

Today there are three instruments in particular of great value in gastroscopy. In the Wolf-Schindler type the lower half of the instrument is completely flexible, but it has a comparatively large diameter. Korbsch's instrument consists of a straight tube into which is inserted a system of prismatic lenses and attached to the tip is a flexible rubber guide which not only facilitates its introduction but adds greatly to the margin of safety. In addition to instruments of these types Chevalier Jackson also uses an open tube gastroscope for the removal of foreign bodies from the stomach and specimens of tissue for histological examination. Such manœuvres require exceptional skill. Introduction of this instrument is carried out through the direct visualization of the œsophagus. A well-equipped gastroscopic service should include all three varieties of instruments.

Our own experience is limited to the use of the Korbsch instrument, and the observations which are described and discussed in this paper are based on 121 consecutive gastroscopic examinations done during the past four years. In nearly all of our patients fluoroscopic examination of the œsophagus and stomach was done and the Ewald stomach tube passed before gastroscopy was performed.

The Preparation of the Patient for Gastroscopy

The examination is usually done in the morning with the patient in the fasting state. In our early examinations we used sedatives such as nembutal or morphine, but this practice was soon discontinued since it was found that we could obtain much better cooperation from the patient without the use of a preliminary sedative. Atropine sulphate causes a certain amount of distress and does not appreciably affect the amount of mucus secreted during the course of examination.

The pharynx and hypopharynx are anæsthetized with a solution of 10 per cent cocaine and adrenalin, or pontocaine 5 per cent, applied with a special curved perforated applicator. To one end of this absorbent cotton is applied, and to the other end a small syringe is attached. The absorbent is kept continually saturated with solution from the syringe and thus the repeated removal of the applicator is avoided. Such preparation occupies about 5 to 10 minutes, and during the interval while local anæsthesia is being produced one has the opportunity of gaining the confidence of the patient and discussing with him the manner in which he can best cooperate.

A large stomach tube is then introduced and the fasting gastric contents are aspirated. Varying quantities of mucus are usually obtained, and in some cases some abnormal gastric discharge. When the aspirated gastric content appears to be abnormal it is sent to the laboratory, where Drs. Fidler and James, working with Prof. Wm. Boyd, have often been able to demonstrate inflammatory or malignant cells in the stained smears. The following instructions are given for the examination of gastric washings:

1. The specimen must be fresh.
2. Centrifuge and pour off the supernatant fluid.
3. Make smears of the debris and fix while wet in Schaudin's fluid (1 part sat. sol. of mercuric chloride, 2 parts of 90 per cent alcohol and 3 per cent acetic acid).
4. Pass the smears through a solution of methyl spirits and iodine.
5. Stain with hæmatoxylin and eosin.
6. Pass through alcohol and mount with Canada Balsam.
7. Make six smears of each specimen. (Positive smears will not be obtained unless the lesion is ulcerating).

Operative Technique

The patient is turned on the left side, with the legs flexed on the abdomen and the head extended on the trunk. This brings the mouth in line with the œsophagus and allows the pylorus to gravitate slightly to the left side. The patient's head rests on a pillow, and an assistant standing at his back helps to support him in this position and to steady his head. The gastroscope is thoroughly lubricated with glycerine and starch and is grasped in the operator's right hand. The index finger of his left hand is placed in the patient's mouth, the epiglottis is pressed forward, and the flexible rubber tip is introduced into the œsophagus. The instrument is passed down the œsophagus, and in the normal case, as a rule, no further difficulty is experienced until the cardio-œsophageal juncture is reached. The acute angula-

Fig. 1

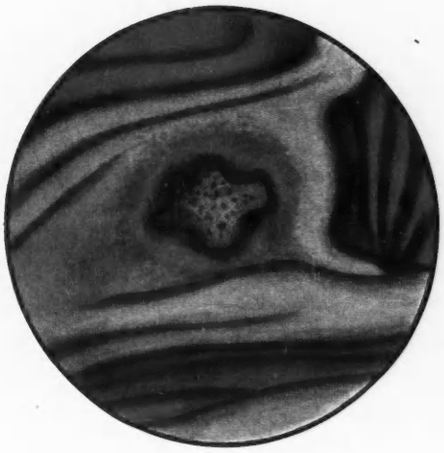

Fig. 2

Fig. 3

Fig. 1.—Gastroscopic view showing ulcer on posterior wall near the lesser curvature.

Fig. 2.—Chronic gastric ulcer.

Fig. 3.—Pyloric antrum showing polyp attached to the posterior surface.

(Illustrations by J. T. Lawson, M.D.)

tion to the left at this site sometimes causes difficulty, but careful rotation of the instrument will usually induce the rubber guide to enter the stomach. It is then passed on down until some slight resistance is encountered. With a comparatively mobile pylorus this should bring the instrument into the pyloric antrum.

The stomach is then inflated with air. This apparently is not a dangerous procedure as excess air escapes around the tube up through the œsophagus. The position of the optic can be readily determined by the position of a small indicator on the eye-piece on the instrument. The lesser curvature is first carefully examined; then the posterior wall, the greater curvature,

Fig. 4.—Pre-pyloric polypus removed at operation. **Fig. 5.**—Skiagram showing the filling defect in the duodenum produced by a pedunculated polypus of the pyloric antrum.

and the anterior surfaces in rotation, as the instrument is withdrawn. Occasionally the pylorus can be seen and the spurting or regurgitation of bile through its lumen observed. The interpretation of any lesions observed is not particularly difficult. The main problem is to satisfy oneself that as much as possible of the gastric mucosa has been subjected to inspection.

THE NORMAL STOMACH FROM GASTROSCOPIC APPEARANCE

Before attempting to interpret pathological changes the gastroscopist should be thoroughly familiar with the normal gastric mucosa in all the parts of the stomach. The normal mucosa as seen through the gastroscope is orange red in colour, has a glistening appearance, and is a deeper red than the sigmoidal mucosa as seen through the open tube sigmoidoscope. The shade of colour varies both with the intensity of the light and the distance of the mucosa from the optic. As the gastroscope is passed from the cardia to the pylorus the mucosa is seen to become lighter in colour.

The surface of the mucosa is thrown into irregular folds or rugæ. For the most part these have a longitudinal direction and are deeper in the pyloric antrum and towards the greater curvature. These rugæ do not appear as long parallel elevations running almost the entire length of the stomach as is sometimes shown in an x-ray rugæ plate, but they have a roughly honeycombed appearance. This apparent discrepancy between the gastroscopic and radiographic appearance is probably due to the fact that in the one instance the stomach is inflated with air and the rugous folds partly ironed out, while in the other the stomach contains a minimum amount of barium and its walls are contracted, throwing the mucosa into deep relief.

At the incisura angularis one sees a fold which seems actually to separate the pyloric antrum from the cardiac end. Schindler has described this and calls it the "musculus sphincter antri".

The beginner should avoid interpreting minute variations from the normal as actual disease. As one might expect, the stomach containing the gastroscope and also air under slight tension is in almost constant motion. This motion is accentuated by the respiratory movements of the diaphragm and the anterior abdominal wall. Some of the finer movements are probably due to the activity of the muscularis mucosæ. With the limited view that is obtained one cannot always determine the exact direction of the peristaltic waves. Physiological reseaches have not shown conclusively that anti-peristalsis does take place. Certainly the expulsion of air around the gastroscope is accompanied by a contraction of the anterior abdominal wall and fixation of the diaphragm. As would be expected, the peri-

staltic activity is much greater in the pyloric antrum than in the fundus; in fact, an area of the fundus may be observed for some time without any movement being seen apart from that due to respiration. One of the most interesting normal phenomena to be observed is the great activity which occurs at the pyloric sphincter. It is constantly opening and closing, and there is an occasional spurt of bile as it is retrojected from the duodenum. The pyloric sphincter was seen in about 55 per cent of our cases.

A striking change is observed when the instrument passes from the cardia to the œsophagus. One sees a crescentic fold at this juncture which appears to be œdematous. The œsophageal mucosa is perfectly smooth and a bright pink as compared to the honeycombed rugæ in the stomach.

Gastroscopy should not be left in the hands of a gastroscopic technician not especially interested in gastro-enterology, for he in his enthusiasm to justify the procedure may be inclined to describe normal variations as minor or major disease processes. As the urologist performs his own cystoscopies so should the gastro-enterologist do his own gastroscopies. He is in the best position to assess the clinical significance of his observations and to correlate his findings with the patient's main complaints. We hope that the unfortunate situation does not arise in this procedure as has occurred in bronchoscopy. The bronchoscopist is usually an eye, ear, nose and throat specialist who knows little or nothing about chest lesions and is entrusted with bronchoscopic examinations and treatment. The fault here does not lie so much with the orificial specialist, who is often a trail-blazer, but with the internist whose abhorrence at inserting his finger or an instrument into any orifice has almost become traditional.

INDICATIONS FOR GASTROSCOPY

Gastroscopic examination is sufficiently and definitely indicated in a patient with upper abdominal symptoms under the following circumstances.— (1) When radiographic findings have been inconclusive or entirely negative in the presence of persistent gastric symptoms. (2) As a pre-operative procedure in all patients who are to be subjected to an exploratory laparotomy of the upper abdomen. (In these cases lesions of the gastric mucosa, *e.g.*, gastritis, polyposis, ulcer, or cancer, could be responsible for the unexplained symptoms.) (3) In all cases where symptoms have recurred following gastro-enterostomy or other gastric surgery. (4) To assist in the differentiation between benign and malignant ulcerations of the stomach when the indications for operation are doubtful. (5) In all cases of unexplained hæmatemesis or melæna. (6) To observe and check the results of treatment in chronic gastritis, or, occasionally, in gastric ulcer. (7) In all cases of so-called gastric neurosis or gastric allergy.

CONTRAINDICATIONS

Gastroscopy should never be done in the presence of:

1. Intrinsic lesions of the œsophagus such as: (*a*) diverticulum; (*b*) primary carcinoma or carcinoma of the stomach invading the œsophagus; (*c*) acute œsphagitis.

2. Lesions such as aortic aneurysms, mediastinal tumours, and angina pectoris.

3. Extreme debility from any cause.

In addition to these definite contraindications, certain conditions render it difficult or even impossible to pass the gastroscope, such as, (1) marked kyphosis; (2) acute angulation of the cardio-œsophageal juncture; (3) large prominent incisor teeth.

During the course of the examination the presence of large amounts of mucus which smears the optic, the constant movement of the stomach produced by the peristalsis, and the movements of the diaphragm and anterior abdominal wall interfere with the ease of the examination to some extent. Nervous patients have not presented any difficulty, but they should be accustomed first to the passage of a large stomach tube.

Once the instrument enters the stomach the dangers are over; there is no chance of perforating the stomach wall, and in our experience no tendency to produce hæmorrhage.

GASTRITIS

At the present time gastritis is classified according to gastroscopic appearance without reference to cause or associated lesions. This, of course, is unfortunate, but it is the best that can be done under the circumstances. Schindler's subdivision into three types, namely, superficial, atrophic, and hypertrophic, is both simple and

practical. Further subdivision into twenty or thirty types as is advocated in some quarters is at the present time confusing and of little practical value.

The superficial type of gastritis has many variations. It may be localized or diffuse, primary, or secondary to other lesions. The mucosa is hyperæmic, with occasional superficial hæmorrhagic spots, and there is a considerable increase in mucus. Such changes may be transient. This fact has been proved by repeated gastroscopic examination. It is conceivable, however, that it may be the forerunner of more severe types of gastritis. We have observed this lesion in association with pyloric obstruction, with peri-anastomotic inflammations, with active ulcer and carcinoma, and, in one case, with advanced bronchietasis. This last patient was suffering from upper abdominal distress, and all other examinations, including visualization of the gall bladder, were negative. We concluded that the swallowing of large amounts of purulent sputum was the etiological factor.

Another very instructive case was that of a man who has been under observation for the past year. In 1922 a posterior gastro-enterostomy was performed for chronic duodenal ulcer. He remained perfectly well until November, 1935, when he suffered a severe gastric hæmorrhage. This was preceded during the previous month by two attacks of mild epigastric pain. He developed a chronic secondary anæmia and a more or less constant epigastric distress, which persisted in spite of time, alkalies, and dietary treatment. In April, 1936, he was investigated. A barium series was negative. No evidence of stoma ulcer was demonstrable, and his gastric acidity was low. There was occult blood in the stool. He had marked dental sepsis, with pus exuding from the gums. Gastroscopic examination revealed a superficial peri-anastomotic inflammation, with pin-point hæmorrhagic spots. The jejunal mucosa was not observed. The stoma was patulous and according to radiographic evidence was functioning well.

The man's teeth were extracted gradually. This was the only important change in his treatment. Two months later he was in perfect health, without pain. He could eat anything and his blood picture was normal. Gastroscopic examination was repeated in October. The peri-anastomotic gastritis had completely disappeared, and in every way his stomach looked perfectly normal.

It is quite possible that the secondary anæmia noted in some of these cases of gastritis with associated marked oral sepsis may be due more to the secondary inflammatory changes in the wall of the stomach itself than to the possible constitutional disturbances produced by the sepsis in the mouth.

In atrophic gastritis the mucosa is characterized by a flat pale surface with prominent blood vessels; it becomes thinned and the rugæ disappear. This type is commonly found in pernicious anæmia and in other marked chronic anæmias. We have not had the opportunity of doing gastroscopies on patients with pernicious anæmia, and have only felt justified in describing the mucosa as atrophic in one of our 121 cases. This patient, aged 68, had a marked hypochromic anæmia of unknown origin. He had achlorhydria. A barium series was negative. There was no blood in the fasting gastric contents or in the stool. Gastroscopic examination revealed atrophy of the mucosa. This patient was treated by his physician with hydrochloric acid, iron and liver extract. This, of course, may have been a primary anæmia in spite of the atypical blood picture. Every clinician with large experience has occasionally had difficulty in differentiating between pernicious anæmia and carcinoma of the stomach, and cases have been reported in the Winnipeg General Hospital of pernicious anæmia which later have developed carcinoma ventriculi. Some of these lesions are probably slowly growing, slowly metastasizing, infiltrating carcinomas, with late ulceration. In the solution of such problems as these the gastroscope should render distinct services to the clinician.

Pentschew, of Sofia, has demonstrated atrophic gastritis following the administration of tellurium. He points out that atrophic changes in the gastric mucosa can be produced by a chemical or toxic agent which circulates in the blood stream.

Hypertrophic gastritis also may present a variety of pictures depending on the severity and the duration of the lesion. The mucosa is thrown into prominent folds, is deep red in colour, nodular, and ragged, with marked œdema, and in the severer cases there are multiple erosions. In such cases the stomach

contains a yellowish blood-stained mucus. We have observed 6 cases with varying degrees of hypertrophic gastritis; in 3 the lesion could be demonstrated by x-ray. Two cases were associated with marked oral sepsis, one with a chronic indolent ulcer situated on the posterior surface of the cardiac end of the stomach. In the other 3 cases no associated lesion was found, and no etiological factor could be demonstrated. It may be significant that 5 of the 6 patients were males. It is believed that this hypertrophy of the mucosa is due to a low-grade inflammation, caused by physical, chemical, or bacterial irritation. The onset is usually insidious, the history goes back over months or years of recurring attacks of epigastric distress and loss of appetite which are readily controlled at first by simple dietary measures; as the lesion progresses the pain and flatulence increase and there are occasionally offensive eructations, loss of appetite, nausea, but rarely vomiting. Attacks of severe pain may occur when the redundant hypertrophic mucosa prolapses through the pylorus. The gastric acidity varies, but as the disease advances hypochlorhydria is the rule. This may be partly due to the neutralizing effect of the alkaline mucus, which is secreted in large amounts. In two of these cases the laboratory reported the finding of inflammatory cells in smears of the centrifuged gastric contents.

The clinical diagnosis is very difficult, but is based on a rather indefinite history of chronic dyspepsia, which may simulate either gastric ulcer, when associated with hyperchlorhydria, or gall-bladder disease, in the presence of hypochlorhydria. In the late stages secondary anæmia and loss of weight may suggest carcinoma. X-ray rugæ plates and gastroscopic examination establish the diagnosis.

The treatment of gastritis depends somewhat upon the stage of the disease. In the early stages simple dietary measures, such as a bland, non-irritating diet, with restriction of fluid taken with meals, may effect a cure. In the severe forms complete rest in bed is essential, daily gastric lavage with soda bicarbonate solution, a bland diet with little fluid during meals, and plenty of water between meals. A powder containing bismuth subnitrate and caroid is useful.

CASE 1

Mrs. P.K., aged 51, reported with flatulent dyspepsia and some vague epigastric distress. She was afraid to eat. There had been 21 pounds loss of weight and some looseness of the stools for one month. These symptoms had developed insidiously over a period of several months. Physical examination was negative, except for marked oral sepsis and some resistance and tenderness in the epigastrium and apparent anæmia. The blood count showed red blood cells 3,600,000; white blood cells 6,500; hæmoglobin 70 per cent. Fasting gastric contents: 44 c.c. of blood-stained mucus; no free hydrochloric acid, even after histamine. The Wassermann reaction was negative. A barium series and visualization of the gall bladder were negative. Sigmoidoscopy was negative. Gastroscopy revealed a markedly congested and hypertrophied gastric mucosa with many superficial hæmorrhagic areas. The diagnosis was chronic hypertrophic gastritis with ulceration. Pathological examination of the aspirated gastric contents demonstrated pieces of gastric mucosa showing marked inflammation. There was no evidence of malignancy.

About six years ago one of us (P.H.T.) performed a posterior gastro-enterostomy on one of the most marked cases of hypertrophic gastritis that has come under our observation, with a very satisfactory relief of symptoms. Of course, in addition to surgery, the patient received thorough post-operative medical treatment. However, this post-operative regimen was followed for only a few weeks, and after leaving the hospital the patient observed no dietary restrictions. This case was under observation before gastroscopy was being done by us and is, therefore, not included in the present series. It is simply of interest in view of the therapeutic result.

At the present time the diagnosis of gastritis is made on insufficient evidence in most instances. One hundred and eight patients were discharged from Winnipeg General Hospital in the past five years with the diagnosis of gastritis. Of these 70 per cent were admitted for alcoholism. Of the remaining 30 per cent the majority were nurses who were admitted for a day or two for transient epigastric distress. An occasional case had been diagnosed by x-ray, and some on gastroscopic evidence. This is probably a representative picture of the state of affairs regarding gastritis in the majority of hospitals on this continent. There is thus a great need of placing the subject of gastritis upon a more scientific basis.

GASTRIC ULCER

Radiography is probably about 95 per cent efficient in demonstrating peptic ulcer. (This percentage may be increased slightly in the most expert hands.) Gastroscopy has then a

rather limited field of usefulness in the diagnosis and management of gastric ulcer. Yet no ulcer service in a large general hospital could be considered complete without facilities for gastroscopic examination being provided in certain selected cases. In our series we have been able to demonstrate gastric ulcers in 5 cases that were reported negative by x-ray examination. In 3 of these cases the radiologist was able, either by reconsidering the original plates or by repeating his examination with mucosal relief plates, to locate the lesion as indicated by gastroscopic examination. Another case was one of a high ulcer associated with marked cardiospasm.

As seen through the gastroscope gastric ulceration can be classified as follows:

1. *Superficial erosions* which are multiple and associated with gastritis and are not related to the problem of chronic peptic ulcer.

2. *Subacute ulcers* which appear in the x-ray plates as a chronic perforating ulcer, and are frequently reported as such. The appearance of a deep crater is, in reality, due to a heaping-up of the mucosa at the edge of the ulcer from inflammatory œdema. This œdematous edge is in reality protruding into the stomach. In this type of ulcer the surrounding mucosa is quite swollen and is inflamed for varying distances. The presence of this intense inflammatory reaction obviously explains the rapid disappearance of the apparent penetration during rigid medical treatment.

3. *Chronic ulcer,* which is characterized by its sharp edge, its greyish but clean base, and, in the majority of cases, perfectly normal gastric mucosa apart from the ulcer (Figs. 1 and 2). Certainly there is nothing in our gastroscopic studies to support the theory that gastritis is associated with malignant or peptic ulceration, according to the gross appearance of the mucosa as seen through the gastroscope. From the reports that emanate from Germany it would seem that marked gastritis is commonly associated with gastric and duodenal ulceration. One of us (C.B.S.) had the opportunity of observing over 65 gastroscopic examinations in the Henning Clinic in Leipsic, and, certainly, the association of gastritis with chronic peptic ulcer was undeniable. This seemed most marked in the pyloric antrum. However, these observations have not been duplicated in our personal series of 121 cases from the surgical or gastroscopic standpoint. In addition to showing the above characteristics, the chronic ulcers are frequently triangular or roughly quadrangular in outline. In some instances we have been able to make a distinction between innocent and malignant ulceration, and with increasing experience we should be able to make this diagnosis with greater assurance.

In several instances we have had the opportunity of observing the progress of an ulcer under active medical treatment. We have been able to demonstrate to Dr. J. C. McMillan, the Radiologist at the Winnipeg General Hospital, the persistence of the lesion for some time after the complete disappearance of all radiographic evidence. The significance of this observation is apparent. Effective medical treatment of gastric ulcer should probably be continued not only until symptoms, radiographic evidence, and occult blood in the stools have disappeared, but until the gastroscope reveals a perfectly healthy mucosa.

CASE 2

Mr. C.S., aged 24, referred in November, 1933, for surgical treatment with a chronic gastric ulcer of six years' duration. He had been on active medical treatment for six months before his admission to hospital, part of this time being spent in bed. He had failed to respond to treatment, and was, therefore, referred for operation. X-ray showed a large crater on the lesser curvature. A sub-acute ulcer was observed through the gastroscope.

The danger of recurrent ulceration in this young patient was considered to be a contraindication to immediate operation, consequently a duodenal tube was passed and allowed to go on into the jejunum. Jejunal feedings were given through the tube and alkalies by mouth. This treatment afforded complete relief from pain in 24 hours. Three and one-half weeks later the x-ray showed a small, but definite, mucosal ulcer, with distinctly more circumscribed and less acute hyperæmia. For this reason the jejunal feedings were continued for another two and a half weeks. A further gastroscopic examination revealed complete healing of the mucosa. Six months later the gastroscopic examination showed a healed mucosa. It is probable that if jejunal feedings had been discontinued when the x-ray findings became negative, early recurrence of symptoms may have resulted. Gastroscopic determination of activity was a distinct advantage in this case. As far as we can determine, this patient has remained well to date.

CASE 3

More recently we have had under our observation a case with two ulcers on the lesser curvature. We explained to the patient that our gastroscopic examination was purely to double-check the appearance of the x-ray during the process of healing. He made no objection to repeated gastroscopic examinations, and we had an excellent opportunity to observe the progress of healing in these ulcers. His previous history had strongly suggested cholecystic disease, and his gastric acidity was normal, but his symptoms were found to be due to the gastric ulcers.

Duodenal or pyloric ulcers cannot be seen through a gastroscope. All surgeons realize how easy it is to overlook a small gastric ulcer at operation when it has not been previously demonstrated by x-ray. Even in cases where the x-ray evidence is definite it still may be difficult to demonstrate the ulcer at operation. Certainly an ulcer which has not been demonstrated by x-ray will not in many instances be discovered by the surgeon. We can say from experience that the small ulcer is more readily detected by gastroscopic examination than by palpation at operation.

In the few stomachs examined after operation we have occasionally observed perianastomotic inflammation but never a stoma ulcer. Stoma ulcers are usually located in the jejunum and cannot be seen through the gastroscope.

POLYPOSIS VENTRICULI

Our experience with polyposis of the stomach is limited to 5 cases in all, 2 of which have been examined gastroscopically. These lesions project into the lumen of the stomach, are usually sessile and purplish in colour. Ulceration and a tendency to bleed (neither of which was observed in our two gastroscoped cases) would suggest malignant change. In addition to confirming the x-ray diagnosis, we can determine the exact extent of the polyposis and the proximity of these lesions to the œsophageal opening. The following case of a single polyp in the pyloric antrum was very interesting and instructive.

CASE 4

Mrs. E.L., aged 58, had some vague upper abdominal symptoms characterized by pain but little indigestion. An x-ray of the stomach showed some filling defect in the pyloric antrum (Fig. 5) which the radiologist thought might be a polypoid tumour. Gastroscopic examination showed a normal gastric mucous membrane. On looking into the pyloric antrum (with the stomach inflated) there was a nodule about 2 cm. in diameter (Fig. 3) having a definite bluish tinge, and it stood out in marked contrast to the rest of the gastric mucosa. It had the appearance of a polypoid adenoma with a wide sessile base, was situated in the pyloric antrum, and was attached to the posterior wall of the stomach At operation this adenoma was excised and proved to be an innocent growth (Fig. 4). The prolapse of the polyp and surrounding mucosa through the pyloric ring undoubtedly produced the atypical attacks of pain. The woman has been completely relieved by operation.

CARCINOMA

Twenty-one of our gastroscopic observations have been on cases of malignant disease. We have seen large polypoid growths projecting into the stomach and also some of the fungating ulcerating type. The malignant ulcer has edges which are irregular, nodular, and dark in colour. The centre has a dirty necrotic base.

We have not had the opportunity of observing a malignant lesion which had not already been shown by x-ray. The early diagnosis of malignancy is one of the most hopeful fields for gastroscopy. We feel that this will be possible when gastroscopic examinations become routine in patients of cancer age, who come to us with chronic indigestion and some loss of appetite or weight. At the present time patients do not present themselves for diagnosis in the early stage of the disease. It is not likely that education of the public regarding the possible serious significance of indigestion in later life will be effective unless we develop some method for earlier positive diagnosis. Our only hope in the future to reduce the high mortality of this disease lies in fluoroscopy, gastroscopy, and early operation. In a few instances gastroscopy has been helpful in determining not only the extent but the operability of a lesion.

A glance at the accompanying statistical chart, shows the present high percentage of cancer deaths due to cancer of the stomach. Such figures present a real challenge to the members of our profession.

CHART I.

DEATHS AND DEATH RATES FROM CANCER PER 100,000 POPULATION

		Stomach and duodenum				*Total deaths (all sites)*	
	Population Manitoba	*Male*	*Female*	*Manitoba Rate*	*Canada Rate*	*Manitoba Rates*	*Canada Rates*
1931	700,139 X	106	57	23.2	22.7	87	92
1932	709,000	151	70	31.2	24.3	94	95
1933	717,000	117	45	22.6	23.9	93	100
1934	726,000	129	51	24.8	24.4	90	98
1935	730,000	128	73	28 3	24.1	106	102

THE DANGERS OF GASTROSCOPY

The dangers of gastroscopy are all related to the passage of the instrument through the œsophagus. At the present time there are so few men doing gastroscopy that the beginner has difficulty in receiving adequate instruction. In our series there have been two accidents, both in patients who were straining a great deal during the passage of the instrument. One patient developed a spontaneous pneumothorax from which he made a complete and unaided recovery. The second was a markedly debilitated man suffering from obstructive jaundice and anæmia. There was inconclusive radiographic evidence suggesting gastric malignancy. The patient refused to cooperate and as there was violent straining the examination was discontinued, while the instrument was still in the œsophagus. That afternoon he experienced a sharp epigastric and left chest pain, which was due to a spontaneous pneumothorax. He later developed a pyopneumothorax which proved fatal in spite of drainage. Autopsy revealed no evidence of injury to the œsophagus and no evidence of mediastinitis.

CONCLUSION

As Chevalier Jackson has said, "Gastroscopy is an addition to and not a substitute for any other method of examination of the stomach". With its aid the gastro-enterologist will be able to distinguish minor but definite changes from the normal, which could not possibly be demonstrated in any other way. One of its greatest contributions has been the placing of the diagnosis of gastritis on a thoroughly scientific basis. For twenty years the diagnosis of chronic gastritis has hardly been accepted as a definite clinical entity, while inflammation of other mucous membranes has been recognized and treated. Accuracy in diagnosis is always increased by a direct visualization of the lesion. In the future it will not be a question of history, test meal, x-ray, and exploratory laparotomy in doubtful gastric cases, but, rather, history, test meal, x-ray and gastroscopy. The grosser defects produced by hypertrophic gastritis, peptic ulcer with a crater, and moderately advanced malignant disease can be readily detected roentgenologically. Certainly the superiority of the gastroscope in demonstrating the finer but often significant pathological changes can hardly be questioned.

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ECHINOCOCCUS ALVEOLARIS*

(WITH THE REPORT OF A CASE)

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ECHINOCOCCUS disease, as everyone knows, is the cystic stage of the tapeworm *Tænia echinococcus*, which passes its adult life in the intestine of the dog. The cystic stage may take one of two forms, the one common, the other extremely rare in many countries infested by the parasite. Human beings become infected by eating unboiled vegetables that have been contaminated by the excreta of dogs that harbour the adult worm.

Echinococcus hydatidiformis, the more common form of the disease, is one in which there is the formation of hydatid cysts. In the intestine the ingested embryo develops into a larva that penetrates the wall of the gut, enters the portal circulation, and is carried to the liver and other organs. The larvæ lodge in the liver, etc., and form cysts which may become as large as a man's head. These cysts consist of a white outer layer and an inner granular or germinal layer. From this inner layer scolices of new worms or daughter endocysts arise by an internal budding process. The cysts are filled with a thin milky fluid.

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Echinococcus alveolaris is the other form of the disease, and differs from the above in that huge cysts are not formed, but those which are produced vary in size from a pin-point up to small hen's eggs. They do not contain daughter cysts and scolices are never present. These cysts are very numerous and arise by an external budding process.

Although the hydatid form of the disease is comparatively common in Canada, Iceland, and Australia, no cases of the alveolar form have been reported in the first two countries and only one case from Australia (Dew,[1] 1931). Of 235 cases of the disease which had been reported up until a few years ago 214 were from central and eastern Europe; the Tyrol and Alps, 30; Switzerland, 32; Bavaria and Wurtenburg, 82; Russia and Siberia, 70. Several cases have been reported from France. In the countries where the alveolar form of the disease is so comparatively common the hydatid form is just slightly more frequent.

ETIOLOGY

There are two main theories regarding the etiology of *Echinococcus hydatidiformis* and *Echinococcus alveolaris*. Some workers believe that these are two different diseases, each caused by a specific type of echinococcus. The other school favours the view that they are different tissue reactions caused by the same parasite. The latter view seems to be the most logical, especially after the work of Dévé[2] and his analysis of three reported cases which show the presence of both types of lesions, and the blending of one type into the other. Dew states "that all echinococcus lesions found in man and animals are essentially the same, and that the pleomorphism they exhibit is due to the occurrence of parasitic variations".

PATHOLOGY

The liver is the most common site of the alveolar form. The lesion consists of a firm tumour made up of many small alveoli or cyst-like spaces which are filled with a mucoid material and surrounded by a fairly dense fibrous stroma. These cysts are rarely larger than 5 to 10 mm. in diameter, and because of their mucoid content were at one time considered as a colloid cancer. In 1855 Virchow recognized their true nature.

The inner granular or germinal layer of these cysts is generally not intact. It forms small buds which extend outside the cyst and follow along tissue planes to form new cysts. The outer layer is a laminated, soft, amyloid-like material which may have a crumpled appearance, so that most cysts are irregular and oval rather than round in outline. Scolex formation does not occur, and reproductive elements are rarely found in the cysts.

Surrounding the budding processes there is an inflammatory reaction like that seen around a foreign body. This reaction is due to a toxin which is liberated. Epithelioid cells, giant cells, and lymphocytes may be numerous, so as closely to resemble the lesions of tuberculosis. The neighbouring blood vessels show an endarteritis, but some may be invaded by the parasitic growth, and metastases may be found in the lungs, spleen, brain, kidneys, etc. The powerful toxin which is liberated by its action on the blood vessels and on the tissues locally frequently causes necrosis, with the formation of a large ragged abscess filled with cholesterol, necrotic material and calcareous matter.

CASE REPORT

The patient was a fisherman, 54 years of age, who came to Canada from Iceland when seven years old, and had resided here ever since. In April, 1928, he consulted Dr. B. J. Brandson because of a large mass in his left hypochondrium which had appeared during the last year, and was accompanied by loss of health and loss of strength. He had always enjoyed good health previously. The mass was globular, smooth-surfaced, and filled the left hypochondrium. A diagnosis of hydatid cyst was made before operation. At operation a large cyst was found replacing practically the whole left lobe of the liver. It was sewn to the anterior abdominal wall, opened, and drained of numerous broken-down daughter cysts and jelly-like material. The cavity was packed with gauze.

The man made a good recovery and remained fairly well for three years, but returned in May, 1935, because of severe upper abdominal pain and a discharging sinus which had been present since his operation in 1928. He had continued to lose weight and for the previous two years had been unable to work. The sinus was excised in May, 1935, but further operative measures were deemed unwise because of hæmorrhage and the poor condition of the patient. Convalescence was satisfactory for about ten days, but then he developed headache, dizziness, dimness of vision and rigidity of the neck. Lumbar puncture revealed a pressure of 18 mm. of mercury, increased globulin, 140 cells per c.mm. (75 per cent polymorphonuclears), and a mid-zone mastic curve on three occasions. The Wassermann reaction was negative. He continued to go downhill and died on July 15, 1935.

Autopsy. — (Only positive findings will be mentioned).

The body was that of a very emaciated male, 54 years of age. The liver weighed 2,125 g. The left lobe was almost completely destroyed and replaced by an old thick-walled abscess containing foul smelling, thick

purulent material, which discharged on to the anterior abdominal wall. Outside this cavity, and extending medially, many small cysts were seen. Numerous cysts were also seen forming large masses in the omentum. They were also present in the walls of the excised sinus. The right lobe of the liver was uninvolved, but was soft and flabby and yellow in colour. The lesions consisted, microscopically, of a central mass of mucoid material surrounded by a fibrous hyaline substance. Many epithelioid and giant cells were present giving a picture similar to that of tuberculosis (Figs. 1 and 2). The

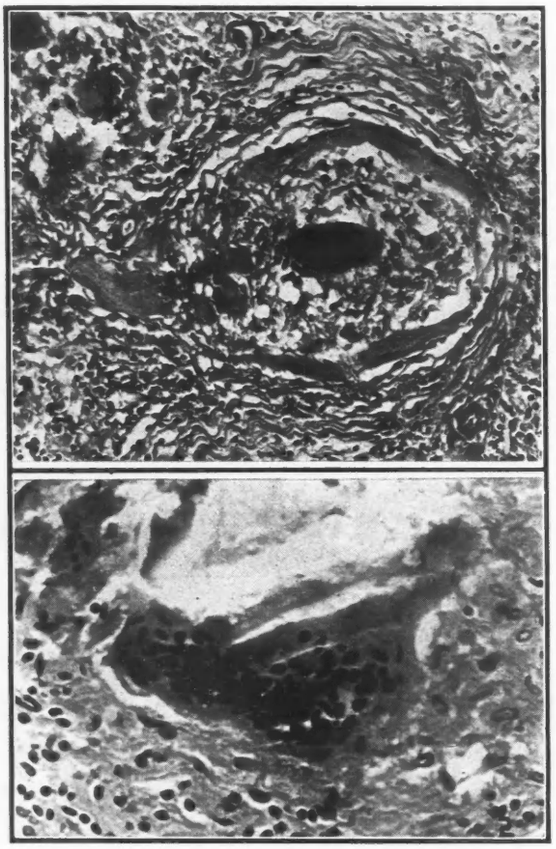

Fig. 1.—Echinococcus alveolaris showing a lesion resembling a tubercle. x 175. **Fig. 2.**—Large multinucleated giant cell. x 325.

N.B.—These magnification numbers are only comparative, as the figures are reduced slightly.

larger cysts were lined by a wavy hyaline material which stained pink with eosin. Within this zone could be seen small fragments of the germinal layer. These two layers were also recognizable in the lesions of microscopic size.

There was congestion of the cerebral vessels, but no lesions in the brain could be found. There were numerous lymphocytes and plasma cells about the vessels in the subarachnoid space.

DISCUSSION

Hydatid disease is comparatively common in Canada, and particularly so in Manitoba on account of the large number of Icelandic emigrants in this province. It is certainly remarkable that not a single case of the alveolar form of the disease has been previously reported in this country, especially if it is granted that the alveolar and hydatid forms are merely different manifestations of the same parasite. The complete absence of the alveolar form in Canada (if we trust to the literature) can be explained in two ways. (1) Cases do occur, but have not been recognized and reported. (2) The assumption that the two forms are different manifestations of the same parasite is erroneous. It is interesting to note that the patient had been absent from Iceland for thirty-six years before the disease showed itself.

SUMMARY

The first-reported case in Canada of *Echinococcus alveolaris.*

We wish to thank Dr. B. J. Brandson whose patient this was for his kind permission to publish this case.

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DERMATITIS FROM DYED AND OTHERWISE TREATED CITRUS FRUITS: REPORT OF TWO CASES.—The practice of colouring citrus fruits by means of dyestuffs, aniline and other substances to give a fully ripened appearance has come into vogue only during the last two to two and one-half years. Traub, Gordon and Van Dyke saw two patients with a dermatitis from handling such treated and dyed fruit. In both instances the dermatitis was caused by the chemically treated or dyed fruit, their patch tests in both patients indicating yellow O B dye as the prime offender. Evidently the dye and chemicals used did not penetrate even to the inside of the peel, as patch tests with the inner surface of the rind gave negative results. Patch tests with the meat of the fruit also were negative. Patch tests with dyed orange skin were repeatedly found to be positive. Patch tests with known undyed and untreated skins (Florida oranges) were negative. In case 1, patch tests with dyed orange peel produced actual blistering of the skin and a severe focal and general reaction. The dyes used on citrus fruits may definitely irritate some skins. These dyes are not general irritants, because the majority of individuals do not react to them. A circumstance of case 1 for which the authors are unable to account on the basis of information supplied them and despite their many repeated tests is the positive reaction to undyed oranges from California.—*J. Am. M. Ass.*, 1937, **108:** 872.

HÆMATEMESIS FOLLOWING APPENDECTOMY*

BY GEORGE H. KITCHEN, M.D.

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POST-OPERATIVE hæmatemesis as a complicating factor of appendicitis is so rare that a cognizance of its gravity and an interpretation of its underlying pathology are demanded whenever this complication arises. For this reason the following case is reported.

CASE REPORT

D.H., male, aged 22, was admitted on April 4, 1935, to the Surgical Wards of St. Luke's Hospital, New York, with the history of severe abdominal pain for thirty hours, and tenderness which already had localized in the right lower quadrant. Nausea and vomiting had followed the onset of pain. The patient took a liberal dose of magnesium sulphate a few hours after the initial symptoms, but it did not afford any relief. Diffuse lower abdominal muscle spasm was present, with the tenderness most marked over McBurney's point. The admission temperature was 100.3° F. The white blood count was 22,000, with 82 per cent polymorphonuclear leucocytes.

A diagnosis of acute appendicitis was made and an immediate operation was undertaken. A gangrenous, perforated, retrocæcal appendix was removed through a McBurney incision, and a rubber dam drain was inserted into the peritoneal cavity. A culture of the purulent peritoneal fluid showed *B. coli communis*. Twenty hours after operation the patient vomited a small quantity of coffee-ground material, to be followed in a few hours by 1,000 c.c. of a similar nature. The vomitus gave a strongly positive guaiac test for blood in each instance. A Levine tube was inserted into the stomach, producing a measure of relief. During the next few days the patient's condition was improved. However, on the sixth day the abdomen became distended; the pulse was rapid and thready, while the temperature rose to 103° F. A transfusion of 500 c.c. of whole blood was given; no further hæmorrhage occurred, but the patient died the next day.

At autopsy three litres of purulent fluid were found in the peritoneal cavity. The coils of small and large intestine were greatly distended. A diffuse thrombophlebitis had involved the omental vessels up to the junction with the gastro-epiploics (Fig. 1). The mucosa of the stomach presented multiple shallow, punched-out erosions. These were situated on the anterior wall of the distal third of the viscus and the posterior wall of the fundus at the cardia. They were from 0.3 to 1.0 cm. in diameter, with a hæmorrhagic, fibrinous base.

Microscopic sections of these lesions show a small, sharply defined denudation of the stomach mucosa which extends almost to the muscularis (Fig. 2). The gastric mucous membrane at the margins of this erosion is remarkably well preserved; the base shows no polymorphonuclear nor other exudative inflammation, such as is found in the base of a chronic ulcer. No granulation tissue has formed, as would occur had the lesion been present for some time, but rather the base consists of œdematous reticular tissue and glandular remnants of the mucosa. The vascular lesion which has undoubtedly produced this erosion appears as a septic thrombus in one vein, adjacent to a very small lymph node under the serosa. This vein is almost entirely filled with polymorphonuclear cells which are adherent to the intima and occlude the lumen. The sinuses of the adjacent node are also filled with polymorphonuclear cells, although the subserous layer is not otherwise infiltrated. This is evidently the path of the pyogenic infection which has finally reached the mucous membrane of the stomach, causing necrosis of a small area, but sufficient to permit of profuse hæmorrhage.

The liver was free from abscesses and the portal vein showed no thrombosis. The remainder of the autopsy findings were unimportant. Death was obviously due to generalized peritonitis.

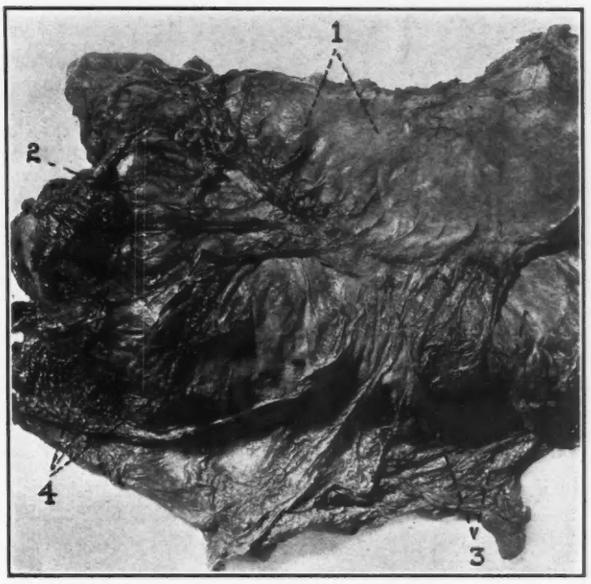

Fig. 1.—(1) Stomach; (2) transverse colon; (3) omentum; (4) thrombosis of the omental vessels.

Von Eiselsberg[16] and Friedrich[5] undertook the study of the relation of omental vessel thromboses to hæmatemesis upon experimental animals. Clamping and tying the omental vessels, they concluded that trauma to the omentum was associated with a retrograde thrombosis of these vessels, which extended to the junction with the gastro-epiploics. They believed that an embolic occlusion was produced by a small portion of the thrombus breaking off. Necrosis of the stomach mucosa followed. Englehardt and Neck repeated this work, and were able to produce the results only under septic conditions. Payr,[10] by introducing dye into the mesenteric veins, was able to produce a thrombosis of the stomach

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veins with mucosal erosions. French workers (Rochard,[12] Broca and Weil,[2] and especially Dieulafoy[3]) described several cases of hæmatemesis following appendectomy, and the last coined the phrase "vomito negro appendiculaire". Blumer[1] took exception to such a term, pointing out that other intra-abdominal operative procedures may have similar complications. Dieulafoy[3] was of the opinion that the gastric mucosal erosions found in patients with ruptured appendix and followed by hæmorrhage were due to the absorption of toxins. He laid emphasis on the presence of icterus and urobilinuria, which appeared in some of his patients, as further evidence of a widespread toxæmia. Purves[11] collected 29 cases of post-operative hæmatemesis, and to these added 11 more. In this series the majority were intra-abdominal operations, but in two instances the operative procedures were on the face and extremities.

Two types of gastro-duodenal lesions have been described to account for this form of hæmatemesis: first, the superficial mucosal erosion which is small, multiple, and rarely extends beyond the muscularis; this group includes by far the major number of reported cases; second, the deep ulcer which is usually single and may perforate into the general peritoneal cavity. McArthur[9] recently described a fatal case of a ruptured appendix which at autopsy also revealed a single perforated peptic ulcer. The duration of this ulcer is not known.

Post-operative hæmatemesis is a very disturbing clinical sign. Purves,[11] in his own series, showed a mortality of 72.5 per cent, while in the collected cases it totalled 69 per cent. The majority of these were associated with peritonitis, but in a few the infection was limited to the appendix. Hæmorrhage usually appears within 48 hours after operation, although Willmoth's[17] patient died on the 45th day after the removal of an inflamed appendix. Hæmorrhages in the fatal cases are usually multiple, while those patients who recover most frequently had a single hæmorrhage. Clinically, the signs of hæmorrhage are rapidly manifested and, added to peritonitis, which is usually present, spell a fatal outcome.

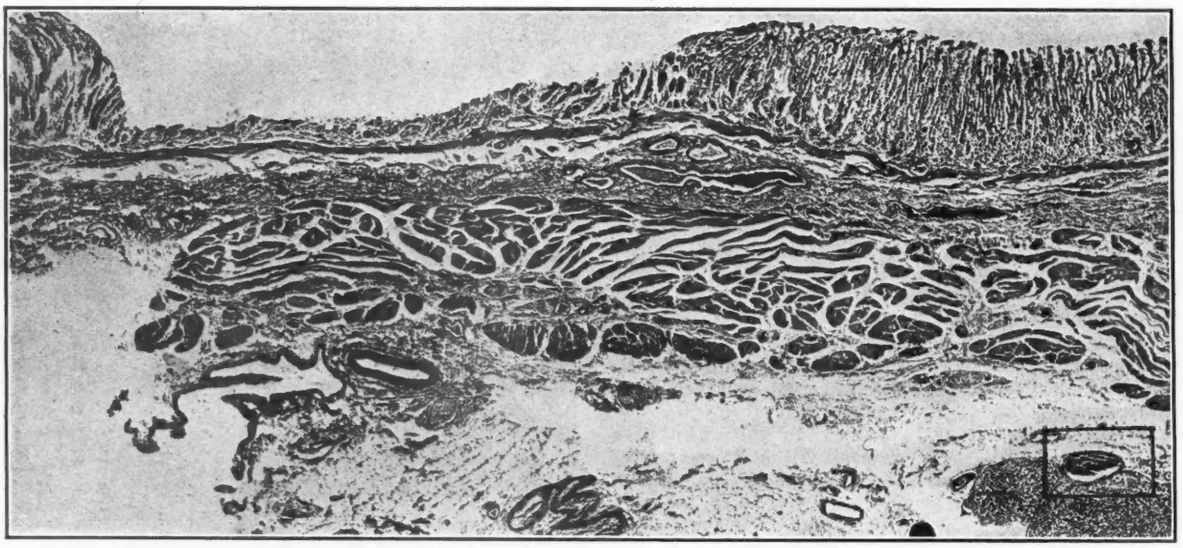

Fig. 2.—Low-power photomicrograph of the stomach wall, showing a mucosal erosion with a septic thrombus in a subserosal vein. The vein is shown at the right in a small rectangle.

SUMMARY

1. Hæmatemesis is a rare complication of acute appendicitis.

2. Thrombosis of the omental vessels is believed to be the cause of this condition.

3. Such a fatal case in a young man is here reported, with autopsy findings.

4. The literature is briefly reviewed.

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BILATERAL CONGENITAL ABSENCE OF THE RADII

BY A. E. HARBESON, B.A., M.D., C.M.

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IN textbooks of surgery absence of the radius is considered to be rare, and only brief references are made to it as a cause of congenital club-hand. Potel[1] quotes Apert and Morisette to the effect that up to 1914 one hundred cases had been recorded, one-third of which were bilateral. In 1917 Lewin[2] reported 14 cases of congenital defects in bones of the extremities among which was one of absence of the radius, complete in the right, partial in the left forearm. In 1912 Whitmann collected over 200 cases with total or partial defect of this bone, (Steindler[3]). Petrides,[4] in 1922, recorded a case of absence of the left radius in a child who died seven days after birth. Alamartine[5] reported an interesting case of a stone mason with absence of both radii and no functional loss.

The foregoing review of the literature would indicate that congenital absence of the radius is rare in orthopædic practice only when bilateral. In a general surgical practice the condition in any degree is rare and usually seen but once in the lifetime of the average surgeon. Therefore the case presented here is considered worth recording.

The etiology of congenital deformities of the extremities is unknown but has given rise to many theories. Among them syphilis, maternal emotional disturbances and trauma during pregnancy have been suggested. The most acceptable, however, is Gegenbaur's, which claims that the suppression of primitive rays during the early stages of embryonic development is the principal cause. A study of the embryology of the upper extremity has been made by Lewis,[6] who found that the arm buds

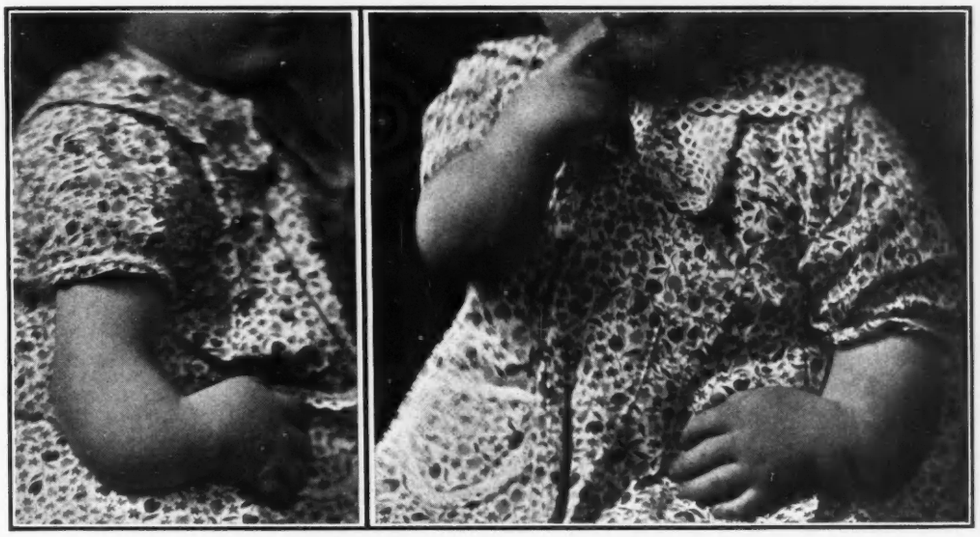

Fig. 1.—Photographs of patient taken March 14, 1936.

appear during the third week of fetal life (4.5 mm.) as a slight swelling of the lower cervical region. By the end of the fifth week the ulna and radius may be differentiated as containing cartilage. The suppression of parts must therefore take place between the third and fourth weeks. At birth the shaft of the radius is completely ossified. The upper and lower epiphyses are ossified from centres which appear at the second and fifth years and join

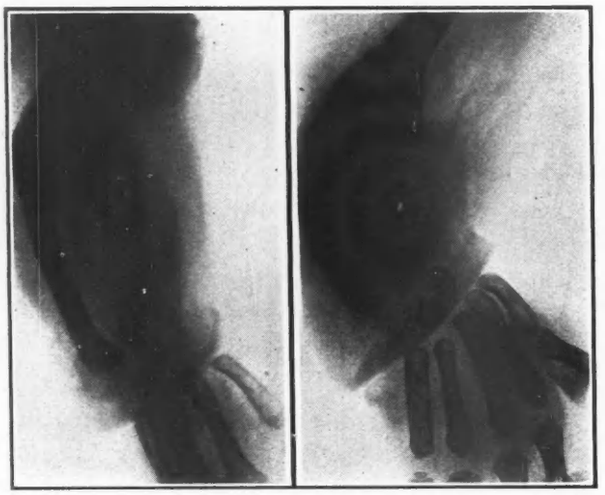

Fig. 2.—X-ray taken March 14, 1936, showing slight ossification in region of proximal end of absent radius in left forearm.

the shaft at the twentieth and eighteenth years, respectively.

Dissections made by Kirmisson and Longet in these congenital cases have shown the muscles on the radial side of the forearm to be almost completely absent. When the ulna was absent the muscles on the ulnar side were defective.

CASE REPORT

A.K. was born at the Hotel-Dieu Hospital, Kingston, on November 30, 1933. Delivery was normal and spontaneous, left occipito-anterior presentation. Weight of child, six pounds, eleven ounces.

Family history.—The parents are alive and well. The patient was the oldest of three. A brother was born a bilateral cryptorchid; an infant sister is normal. There was no history of lues, tuberculosis, or alcoholism.

Examination revealed a well-nourished, healthy baby girl, aged two and a half years. She had bilateral club-hands, with radio-palmar deviation of the hands from the normal axis. The forearms were shortened and were held at right-angled flexion. They were pronated and there was only slight movement of either elbow-joint. Only one bone was palpable in each forearm. There were five digits on each hand. The thumbs were apparently fully developed, but were functionless. The flexed position of the forearms was maintained even when sleeping. In all other respects the child appeared normal.

An x-ray on December 2, 1933, showed complete absence of both radii. The ulnæ were bowed, with the convexity posterior. The metacarpals were all present.

An x-ray on March 14, 1936, showed little change. Ossification of the hamate and capitate bones was evident. There was slight ossification in the region of the head of the radius of the left forearm only.

In this case there is nothing in the way of treatment that would benefit the patient at the present time. After ossification of the carpal bones it may be possible to remedy the condition by means of one of three methods devised, *viz.*, Bardenheur's,[7] in which the ulna is split, and the carpus inserted between the two ends; McCurdy's,[8] in which the distal edge of the ulna is cut through and the proximal end inserted into the radial side of the carpus; and Albee's,[9] in which a tibial graft is transplanted into the junction of the middle and inferior thirds of the ulna and into the navicular bone. Although the immediate results are often good following these operative measures, the final outcome is sometimes disappointing. If the extremity functions fairly well it is better to leave it alone.

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AURICULAR FIBRILLATION: ITS INFLUENCE ON COURSE OF HYPERTENSIVE HEART DISEASE.—Auricular fibrillation, the most common form of arrhythmia in hypertensive heart disease, occurred in 158 (25.3 per cent) of 623 patients with hypertension analyzed by Nathan Flaxman. It definitely influenced the course of the disease in forty-four patients (27.8 per cent) in whom the rapid irregularity preceded and precipitated the congestive heart failure and led to an early death from this cause within one month after the onset in eight (18.1 per cent) of the forty-four patients. When the auricular fibrillation occurred after congestive heart failure had been present from one month to several years, it had no apparent influence on the course of the disease except in relation to the cause of death and the comparative absence of additional occurrences common to appear in hypertensive patients.—*J. Am. M. Ass.*, 1937, **108**: 797.

PRIMARY SARCOMA OF THE UVEAL TRACT*

(AN ANALYSIS OF TWENTY-SEVEN CASES)

BY S. HANFORD McKEE, B.A., M.D.

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FOR practical clinical purposes there are two kinds of primary intraocular tumours: (1) sarcomata of the uveal tract, which occur as a rule in adult life, and destroy by metastasis or extension; and (2) retinoblastomata, which are found generally in infants, have a familial tendency, and destroy by local extension and also by metastasis. As a rarity one also finds metastatic carcinoma and metastatic sarcoma of the choroid. In the uveal tract sarcomata appear much oftener in the choroid than in the iris or ciliary body. Fuchs[1] puts 6 per cent of the cases in the iris, 9 per cent in the ciliary body, and 85 per cent in the choroid.

Malignant melanomata are relatively rare. According to Fuchs[2] they occur in about 0.066 per cent of all eye cases. Stallard[3] states that during 1925 to 1931 at Moorfields, sarcoma of the choroid was found in 1 of every 4,000 patients attending the out-patient clinic. They occur mostly between the fortieth and sixtieth years, though in rare instances they have been found in children. Males and females are affected about equally. Rarely does this disease present itself in both eyes.

The earliest clear reference to intraocular sarcomata arose from a discussion upon the subject of pigmentation. Laennec[4] in 1819 first drew attention to this peculiarity as a means of classification and nomenclature. Lawrence,[5] in 1845, Tavignot,[6] in 1853, and others regarded them as malignant, while Sichel,[7] in 1851, distinguished between benign and malignant melanosis. Virchow,[8] in 1863, distinguished between simple melanoma, melanosarcoma, and melanocarcinoma, and laid stress upon the fact that pigmentation was of secondary importance, the real test of malignancy being the histological structure. In 1868, v. Graefe and Knapp,[9] simultaneously published clear and accurate accounts of the clinical course of these tumours. In 1931 Callender[10] reported in detail the examination of 111 cases which had been sent to the Washington Museum. He described four or five specific types into which he believed all primary malignant uveal neoplasms could be classified. In 1933 von Hippel,[11] from the Göttingen Clinic, reported a series of 118 cases. Evidence of metastases in this series was 38 per cent, the majority occurring within five years. He believes this figure is too small and that 50 per cent would be approximately correct. In 1934, Callender[12] reported on a new series of 196 cases, which included his previous 111. Last year Terry and Johns,[13] of Boston, gave a preliminary statistical study of the cases from the Pathological Laboratory of the Massachussets Eye and Ear Infirmary.

Sarcoma of the choroid appears in two well known forms—circumscribed and diffuse. In the latter the choroid is diffusely thickened in its entirety. This form is rare.

The clinical course of sarcoma of the choroid may be divided into four stages. (1) The first manifests itself by detachment of the retina at the site of the tumour. Patients may notice a disturbance in the visual field, and examination with the ophthalmoscope now shows at the source of the tumour that the vessels are engorged, the veins dilated, and the colour of the retina grey or greyish blue. The retinal detachment is generally sharply defined, and one can often discern the dark tumour mass upon which the superficial tortuous vessels are visible. At this stage the differential diagnosis is most important, especially now when so much operative attention is being given to detachment of the retina. In patients between the fortieth and sixtieth years spontaneous detachment of the retina in a non-myopic eye should be held in great suspicion. (2) The second stage is called the glaucomatous or inflammatory, where, with increase in the size of the intraocular growth, the tension within the globe is raised. (3) The third stage is marked by rupture of the tumour through the

* Read at the Meeting of the American Academy of Ophthalmology and Otolaryngology, New York, N.Y., September 29, 1936.

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eyeball. (4) The fourth stage is that of metastasis, when secondary nodes usually appear, first in the liver, then in the lung and other organs. These four stages can last from two to four years, and the first two are considerably the longer.

Sarcoma of the choroid belongs to the most malignant of eye diseases. The prognosis in a certain percentage is absolutely unfavourable for the life of the patient if the eye is not removed early. Even then the prognosis is by no means to be regarded as perfectly favourable. Local recurrences in the orbit and metastasis also may develop after the removal of the eye. Sarcoma of the uveal tract is hence to be regarded as one of the most malignant of diseases and one which in very many cases ends in death.

The origin of the essential pigment of the eye-melanin may have an important pathological bearing on the nature of intraocular tumours. Bloch isolated from the embryo of the broad bean 3.4 dehydroxyphenylaline, a substance which he called "dopa" and showed that it was readily changed to melanin by an oxidase found in the cells of pigmented tissues. When this substance is added to epidermal cells of skin in frozen formalin-fixed sections, granules of melanin are formed (the "dopa" reaction).

Melanin is closely allied to adrenaline, and it is probable that the two substances are derived from the same precursors and form alternative end-products in metabolism. When the cells are stimulated to proliferate so that there is an increase of the oxidative ferment a pigmented tumour is the result, or a generalized melanomatosis, as in Addison's disease. It would seem from clinical observations that in the eye melanogenesis is a function both of epiblastic and mesoblastic tissues, and recent chemical work seems to substantiate this deduction. Chemistry falls in line with clinical observations, and so far as the eye is concerned it seems clear that pigmentation may be either ectodermal or mesodermal, so that malignant melanomata may be either carcinomatous or sarcomatous. In this respect Terry and Johns[13] state, "In recent years grave doubts have arisen as to the mesoblastic origin of the class of primary malignant uveal neoplasms called sarcomata. In fact Masson and others have presented strong evidence that is indicative of ectodermal origin".

The question of pigmentation is an intriguing one. Under normal conditions pigment in the body occurs in the rete malpighii of the skin, in the eye, in muscle and in fat. Pigment formed under pathological conditions may be derived from the blood (hæmatogenous), from bile (hepatogenous) may be elaborated by various cells after the analogy of the normal pigment-metabolic, or may be introduced into the body from without by drugs, as in tattooing or by inhalation (extraneous). Melanin occurs normally as the colouring matter, of the hair, the choroid of the eye, of the skin, and in the pigment matter of many lower animals. Pathologically, melanin occurs chiefly as the result of an excessive production of this pigment by cells normally forming it, as in freckles, melanotic tumours and Addison's disease. The manner of this excessive production by melanotic tumours is by no means solved, nor can the production of melanin on normal melanin-bearing tissue be considered as satisfactorily settled. Quoting Terry and Johns, there are four possible sources of pigment in uveal sarcomata. (1) The true melanotic pigment, produced or laid down in and between the tumour cells; (2) normal chromatophore pigment of the uveal tract engulfed by the growth of the tumour; (3) pigment epithelium of the retina that either has been engulfed in the tumour or has grown into that tumour; (4) blood pigment from hæmorrhages or stagnant blood spaces that have been invaded or engulfed by the tumour.

In a number of the less pigmented tumours of our series pigment was found to be denser at the base (it is sometimes quite possible to note this basal collection of pigment with the ophthalmoscope). The age and nutrition of the parts may be more important factors favouring melanin-production than the cell type of the tumour.

In 1931, Major Callender[10] described four or perhaps five specific types into which all primary malignant uveal neoplasms can be classified.

1. *The spindle-cell type.*—Sheets, whorls, and other irregular arrangement of spindle-shaped cells with long oval nuclei. The ends of the cell appear to terminate in fibres, so that the cells resemble fibroblasts. There is no argentophile reticulum except that accompanying

the nutrient vessels. These spindle-cell tumours can be separated into two divisions by the nuclear characteristics.

Sub-type A.—The nucleus has a delicate reticular structure in which the nucleolar material is not well defined. These tumours are usually fairly heavily pigmented.

Sub-type B.—The cell has a sharply defined deeply stained small round nucleolus, usually situated near the centre of the nucleus in a rather coarse nuclear network, and is usually lightly pigmented.

2. *The fascicular type.*—The cells of this type are elongated, sometimes forming fibre-like structures, but rounded and polygonal shapes are seen. The characteristic nucleus is oval, has a distinct nucleolus, and closely resembles the nucleus of spindle-cell sub-type B. The majority of the cells are arranged in columns or fasciculi, the long axis of the cell being at right angles to that of the column. The cells radiate about the centre of the column in a palisade arrangement, the centre being a lymphatic or capillary blood vessel. Pigmentation is usually scanty.

3. *The epithelioid type.*—Polygonal cells, usually of relatively large size, though there is considerable variation in both size and shape. The nucleus is large, round or somewhat oval, and the nucleolus is distinct. This type varies greatly in the degree of pigmentation.

4. *The mixed-cell type.*—Irregular mixtures of spindle and epithelioid types of cells, with occasional areas of the fascicular type. These tumours are very heavily pigmented.

Terry and Johns[13] state that from the standpoint of immaturity of cell forms one would consider the epitheloid type to be the most malignant, but Callender actually found the mixed to be the most malignant. In his report of 196 cases classified in the above 5 groups:

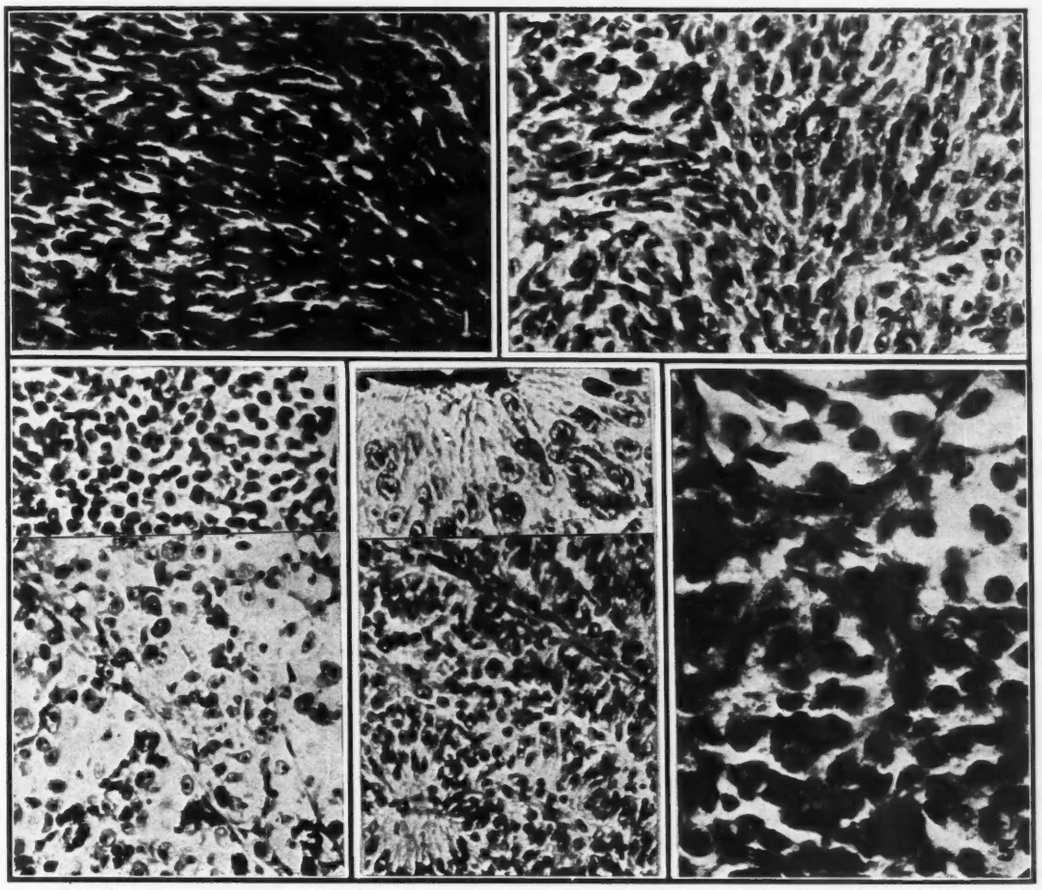

Fig. 1.—Spindle-cell, sub-type A. **Fig. 2.**—Spindle-cell, sub-type B (from the article by Callender, 1931). **Fig. 3.**—The epithelioid type (from the article by Callender, 1931). **Fig. 4.**—The fascicular type (from the article by Callender, 1931). **Fig. 5.**—Orbital recurrence from the epithelioid type.

There were 36.64 per cent of deaths in the mixed type; 25.00 per cent of deaths in the fascicular type; 21.42 per cent of deaths in the epithelioid type; only 6.81 per cent of deaths in 44 cases of spindle-cell B. No deaths in 25 cases of spindle-cell sub-type A.

Since the war period 27 cases of primary sarcoma of the eye have come under our observation. Fifteen eyes were from males; 12 were from females. The age incidence was as follows:

20 to 39 years	.....	6 cases
40 " 49 "	.....	6 "
50 " 59 "	.....	7 "
60 " 69 "	.....	6 "
70 years and over	.....	2 "

In 16 cases it was a history of visual disturbance that brought the patients to consultation. The last 4 cases which only recently came to the

outdoor clinic had consulted the optical departments of departmental stores, without, of course, much relief. In 4 cases there was a definite history of previous injury to the eye by contusion. The clinical diagnosis was made in all cases except two where diagnosis was impossible because of cataract and glaucoma which prevented a view of the interior of the globe. Separation of the retina is the most common complication of malignant melanoma and was observed in all our choroidal cases except the two just noted.

Orbital recurrence has been rarely noted and in our series was present in only 2 cases. Terry and Johns suggest that the orbit may be an unfertile field for tumours, and that Tenon's capsule may hold the tumour in check long enough at least for metastasis already existing or metastasis from the orbital recurrence to kill the patient before local recurrence manifests itself.

The liver is definitely the most fertile field for metastasis, and while most metastases are blood-borne, metastasis by the cervical glands does occur. We were fortunate in obtaining post-mortems in two cases, details of which will show how extensive the metastasis generally is.

CASE 1

A.L., a male of 50 years, was sent in to hospital in February, 1924, with a diagnosis of detachment of the left retina and sarcoma of the choroid. The eye was enucleated. He was readmitted to the Medical Ward in March, 1925, and died the following April. The autopsy report in part was as follows. Melanotic metastasis in the dura mater, brain, occipital bone, clavicle, 3rd rib, diaphragm, pericardium, peritoneum, mesenteric and mediastinal lymph nodes, epicardium, myocardium, liver, pancreas, left kidney, adrenals, bladder mucosa, great omentum, thyroid gland, 11th thoracic vertebra. The liver showed extensive metastasis and extended to the level of the anterior superior spine in the mid-centre line.

CASE 2

J.M., aged 52 years, came to post-mortem in February last from sarcoma of the left choroid with metastasis to the abdominal wall and intestinal obstruction. There were found metastases to the hilus of the left lung, peritoneum, intestines, mesentery, liver, kidneys, adrenals, perinephritic tissue, pancreas, bladder, left femoral glands, and the subcutaneous tissue of the abdomen.

CASE 3

Miss C., aged 35 years, complained of visual disturbance. A detachment of the retina was found and enucleation advised. This was in 1929. The patient died from multiple metastasis in 1931.

CASE 4

Mr. P., aged 46 years, had a similar history. His eye was enucleated in 1928; he died in 1931.

In the first two cases the cell structure was classified as epithelioid, the third fascicular, and the fourth mixed. Compare these with the following.

Mr. D., aged 62 years, had his eye enucleated in 1926. He is alive and well. Mr. D., aged 34 years, had his eye removed in 1927. He is alive and well. Miss N., aged 21 years, had her eye removed in 1928. She is alive and well. Mrs. U., aged 72 years, had her eye removed in 1932. She is alive and well. Mrs. R., aged 64 years, had her eye removed in 1931. She is alive and well.

The cell structure in all this second group of cases belongs to the spindle-cell sub-type A group.

In our series of 27 cases there were 26 tumours in the choroid and 1 in the ciliary body. Pigment, graded 1 to 4, was present in every case in varying degree.

The cellular classification has been altered from time to time during prolonged study. At present it stands as follows:

Mixed cell type	2 cases
Epithelioid cell type	7 cases
Fascicular cell type	4 cases
Spindle-cell sub-type A	14 cases

This result means, according to Callender's last report, that in a little over 50 per cent of our cases the prognosis should have been, and is in fact, excellent. Likewise in the other 50 per cent the prognosis is bad. You may remember that in von Hippel's figures he suggested that metastasis in 50 per cent would be approximately correct.

CONCLUSIONS

In recent years grave doubts have arisen as to the mesoblastic origin of the class of primary malignant uveal neoplasms called sarcomata. It is believed by some that they arise from the epithelial cells of the retina, whether dispersed in the choroid or as misplaced elements. A name should also indicate in these tumours some relation to melanotic pigment, since there can be little doubt that everyone of these tumours has a tendency to produce melanin. This was present in every one of the 27 cases of our series. Terry and Johns have on these grounds suggested the term "malignant melanoma" in place of "uveal sarcoma". They concluded from their study that freedom from metastasis for the usual five-year period was not a sufficient interval in which to determine the malignancy of malignant melanoma. Other

observers have noted metastasis as late as twenty-four years after enucleation. In one of our cases death occurred from multiple metastasis nine years after enucleation.

Separation of the retina is the most common complication of malignant melanoma. It was observed in all our cases except two. The disturbance of vision thus caused was what brought 16 of our 27 cases to consultation. May I remind you that a serous detachment of the retina may be the primary symptom also in metastatic carcinoma of the choroid. I repeat that in a patient over the fortieth year a spontaneous detachment of the retina in a non-myopic eye should be held in the greatest suspicion.

Malignant melanoma can be graded according to cellular morphology, although the value of this grading can only be determined by the study of many more cases, with an intense follow-up extending over a comparatively long period. It is imperative, if we wish to correctly classify those tumours by the cell type, that serial sections of the whole globe be made. Otherwise the chance of error is great.

Callender and Wilder,[14] in their paper on the prognostic significance of argyrophil fibres, conclude as follows.

1. "Classification according to fibre content is most valuable for subdividing melanomata of mixed-cell type into groups of relative malignancy, and deaths in the spindle-cell group appear to be explained by the presence of areas containing no argyrophil fibres.

2. "Classification according to fibre content in conjunction with cell typing affords an aid in prognosis, an abundance of fibre production indicating a more favourable prognosis and a decrease in fibre production denoting an increase in malignancy.

3. "The original classification by cell type, based on hæmatoxylin and eosin staining, still holds, but a more accurate prognosis is made possible by additional classification according to fibre content."

While the duration of observation in this small series has been insufficient to allow definite conclusions to be drawn, unquestionably the follow-up of these cases tends to show that the spindle-cell sub-type A is definitely the most benign of the Callender types. To date no case of spindle-cell sub-type A in his series has given rise to metastasis, while the mixed-cell and other types have given rise to a large percentage of metastases and death. This study leads us to believe that the Callender cellular classification gives us very definite information as regards prognosis in this dread class of malignant disease. Surely a wonderful step forward, though we must bear in mind that it is only a step.

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MULTIPLE FRACTURES OF INFANCY. — R. Dubois and E. Helaers-Balle enumerate the many conditions which may be responsible for multiple fractures in children, and divide them into those which may be acquired and those which are of congenital origin. In the former group rickets and osteomalacia are included, while heredity may be responsible for many cases of bone fragility such as osteopsathyrosis, periosteal dysphasia, osteogenesis imperfecta, or osteopetrosis. Rickets of infancy presents many characteristic signs such as epiphyseal enlargement, curvature of the bones, persistence of the fontanelles and sutures, craniotabes, loss of muscle tone, and anæmia. Radiography shows characteristic changes in the bones. Osteomalacia is characterized by bony softening, accompanied by pain and frequent fractures. Radiography reveals a marked decalcification of the skeleton; generalized osteoporosis is present with hypocalcæmia. The condition is rare in early infancy, and seldom occurs before the age of 2 years. Osteopsathyrosis shows an extraordinary disposition to fracture. The break, which is usually seen at the neck of the long bones, is caused by the slightest injury, and the bone snaps suddenly like a piece of glass. Healing is usually rapid. The condition is associated with blue sclerotics; it usually becomes manifest about the age of 2 years, and often disappears after puberty. An x-ray examination may show a thinning of the cortex of the diaphysis of the long bones and an unusual transparency. Osteopetrosis is a congenital condition, characterized by an increase in density in all the bones and a tendency to fracture. In most cases there is anæmia, enlargement of the liver, spleen, and lymphatic glands, atrophy of the optic nerve, and sometimes hydrocephalus. Union of fractures in these cases is rapid, usually with excess of callus formation.—*Le Scalpel*, February 22, 1936, p. 225. Abs. *Brit. M. J*

THE TREND OF DIABETES IN SASKATCHEWAN, 1905 TO 1934*

By LILLIAN A. CHASE, M.B.

Regina

THE North-west Territories were divided in 1905 when Saskatchewan became a province. Thus we have an opportunity of studying the trend of diabetes in a province whose settlement by white men began just over fifty years ago. In 1905 the population of Saskatchewan was 236,000; by 1933 it had increased to 951,000. Seventy per cent of the population is rural.

Racial origins are varied. Approximately one-third of the population were born outside of Canada (1931 census). The immigrants came from Great Britain, from the United States, and from Europe. Germany, Russia, Austria, Poland and the Scandinavian countries have contributed settlers. There are very few Italians, Spaniards, Japanese or negroes, but there are a number of Chinese. The native Indian population is estimated to be 15,000; there has been a substantial increase in their numbers in the past ten years.

Temperature variations are extreme, from 56° below zero in the winter to 104° in the summer, giving a range of 160°.

A great deal of popular interest and disappointment has been aroused by the publication of statistics which show that the death rate from diabetes has not ceased its upward trend since the discovery of insulin. That it continues to increase thirteen years after the insulin has been in general use is of interest to the physician and to the statistician. It will be shown from an analysis of statistics that little assurance can be given that the rate will drop in this province. Diabetes ranks eighteenth in the list of the causes of death in Saskatchewan.

Careful studies have been made on the trend and incidence of diabetes by Joslin, Dublin and Marks,[1] and by Ross and McKinnon,[2] for Ontario. All students of the problem find the same tendencies. The general death rate from diabetes is rising. The rate for those under fifty is falling, but the rate for those over fifty is increasing faster than the rate of those under fifty is falling. The rate for women is increasing faster than that for men.

A country with an average young population has a low diabetic death rate. The lower the income, the higher the birth rate. Raymond Pearl says that this class differential fertility is always present in society. In 1934 the city of Regina, with a population of 60,000, had 18 per cent of the population on relief. There were 918 births to Regina residents; 286 of these babies were born to those on relief; the birth rate of those on relief was 26 per 1,000 and of those not on relief, 13 per 1,000. So in countries with a low per capita income there is a high birth rate, with a resulting low average population age. The following countries have a higher birth rate than Saskatchewan: Greece, Hungary, Italy, Lithuania, Poland, Roumania and Spain. All of these countries have a low per capita income. With their high birth rate goes a low diabetic death rate. Italy in 1931 had a rate of 8.2; Spain had a rate of 8. In the more prosperous northern countries, with lower birth rates and higher average population age, there are higher diabetic death rates. Norway in 1930 had a rate of 11.6, and Holland in 1930 had a rate of 17.6.

In Chart 1 we see the rising rate in Saskatchewan. Reasons for this rise are as follows. The infant mortality is falling, the death rate from tuberculosis is falling, leaving more people to grow to maturity and to develop diabetes in the fifth decade, if not earlier. The average population age is increasing, because people who immigrated as youths are growing older and immigration has ceased. In spite of falling incomes the birth rate is following the downward trend observed in all parts of the world where births are not encouraged by subsidy or by propaganda.

Other reasons why the death rate from diabetes does not improve are: the carelessness of the young diabetic, who feels that he can show sugar without danger, because insulin will always bring him back; but some intercurrent infection often lurks in his system, causing death. Theoretically, the young diabetic should live to old age; it is not the present lack of scientific knowledge that causes him to die, but his own self-indulgence. Knowledge of modern

* A grant from the Banting Foundation has made this study possible.

diabetic treatment has been available since 1922, but this knowledge has not completely penetrated the medical profession. Definite knowledge of how to treat the diabetic is easily available, but is not always used. Then there are the fake remedies that promise freedom from glycosuria without the use of insulin. They run the gamut from the use of the Abrams' machine to pills.

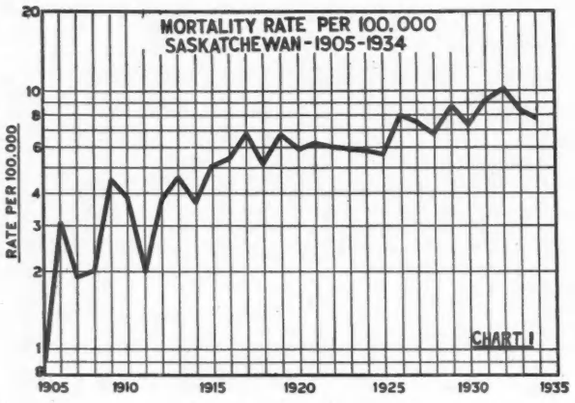
Joslin *et al.*[1] have stated that, "whenever and wherever the conditions of life are easy, food abundant and relatively cheap over long periods, and when large numbers of individuals become accustomed to partake of food in excess of their requirements for the expenditure of energy, the frequent development of overweight and diabetes is favoured. The United States has the highest diabetic death rate in the world. Relatively high rates prevail in most of the British colonies. In Europe the rates in the northern countries are higher than in the south and east, which means that they are higher among the Germanic groups than among the Latins and Slavs. It is precisely in countries where diabetes is most frequent that the level of per capita income is high, and it is reasonable to assume that the level of nutrition corresponds with the per capita income."

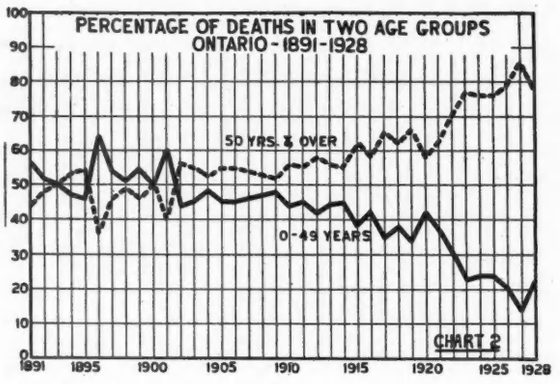
Since 1930 the income of the people of Saskatchewan has decreased in a rapid and spectacular fashion. But the people do not lose weight because of poor diet; when they are short of money they eat an unbalanced high carbohydrate diet, because carbohydrate is the cheapest food available. Flour and cereals yield more calories to the dollar than any other foodstuff; low-calorie diets, with their vegetables and proteins and fresh fruits, are expensive. The following conditions help to produce the low rate in Saskatchewan: a young population (23 per cent of the population is under ten years of age); an agricultural population (70 per cent of the population is rural); no city is over 60,000; a high birth rate, 21.2 per 1,000 (1933); a low per capita income (if this be a factor; 20 or 30 per cent of the population have been on relief at some time during the last five years). The only province in Canada which has a lower rate is Alberta (7.5), a province in most respects similar to Saskatchewan. In spite of the present favourable rate we have no reason to think it will not become worse. Immigration has ceased; generally speaking, immigration brings in the young and healthy. The birth rate is falling, in spite of falling incomes. The rate of natural increase was 20.2 in the years 1921 to 1925; in 1933 it was 14.9. Therefore the average age of the population is increasing; each year will see an increase in the proportion of those over fifty years.

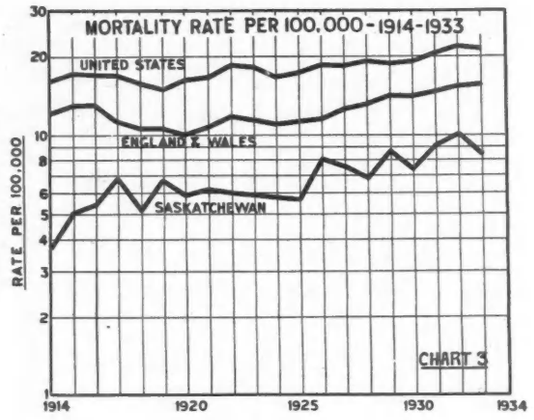
The general death rates from diabetes are shown in Chart 1. The lowest rate recorded for a complete year was 1.9 in 1907, the highest was 10.1 in 1932. The diagram shows a general upward trend marked by small irregularities. The rate of increase was more rapid in the first twelve years than it has been in the last eighteen years. The rates for Saskatchewan from 1905 to 1933 are similar to those of Ontario from 1880 to 1910. In 1880 Ontario was a new unsettled province, just as Saskatchewan was in 1905. The Saskatchewan diabetic mortality curve is similar to that of Ontario, as shown in Chart 2, but it begins twenty-five years later, just as the settlement of Saskatchewan began later than that of Ontario. From 1910 to 1928, eighteen years, Ontario's rate increased 4.1 points, a slow but steady increase. We predict that in 1952 the Saskatchewan diabetic death rate will be about 13. There is no reason to suppose that it will decrease.

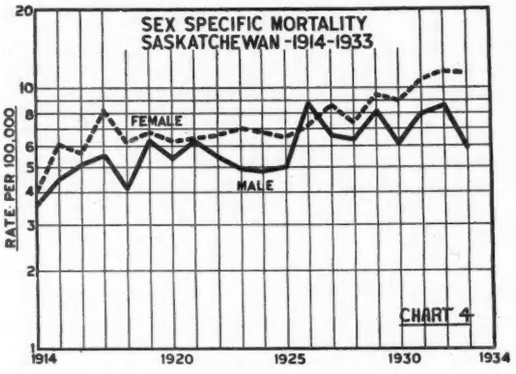
In Chart 3 Saskatchewan's rate is compared to that of England and Wales and of the United States. Since 1915 Saskatchewan's rate has risen from 3.7 to 8.4; the rate of England and Wales from 12 plus to 15 plus; that of the United States from 16 plus to 20 plus. But an increase from 3.7 to 8.4 is twice as rapid a rate of increase as from 12 to 15 and from 16 to 20; it is similar to Ontario's rapid increase from 4.1 in 1886 to 9.5 in 1905.

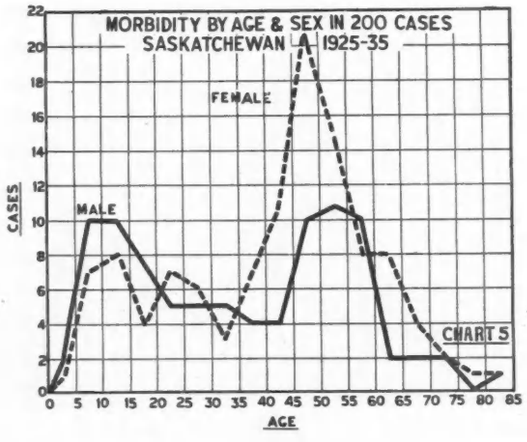
The factor of sex.—The sex-specific death rates for 19 years are shown in Chart 4. With the exception of two years, 1921 and 1926, the rates for females have been definitely higher than for males. All over the world this tendency for the feminine rate to increase has been noted. In a subsequent paper the influence of pregnancy on the development of diabetes will be studied. Some students[1] have thought that the increase in the feminine rate is due to the greater ease of the life of the female on account of improved economic conditions. This reason does not apply to Saskatchewan. The life of the female here is hard. Since 70 per cent of the population is rural that means that they have no modern conveniences, no running water. Anyone who has done housework where every drop of water has to be carried in and carried out, and lifted here and there cannot be said to have an easy life. The country woman finds it

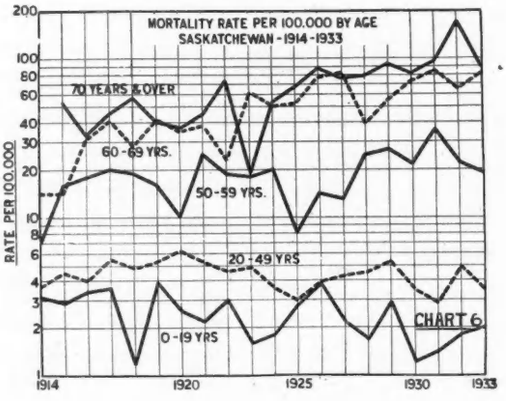


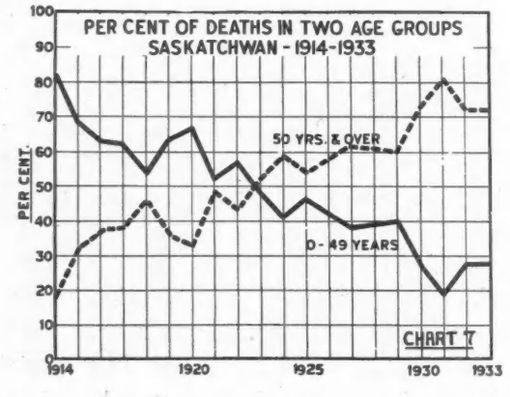


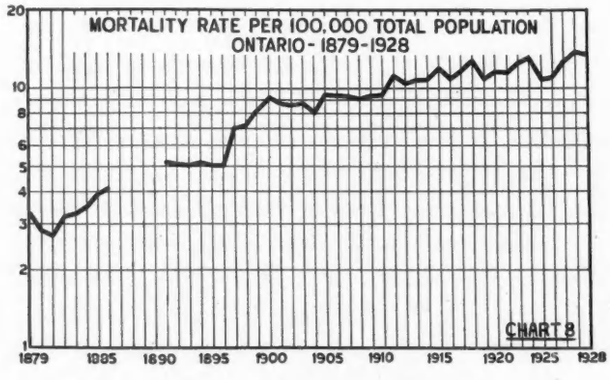












Charts 2 and 8 are reproduced by permission from the article by Ross and McKinnon.[2]

hard to get any outdoor exercise for six months in the winter, and lack of outdoor exercise causes the metabolism to slow down and the weight to increase. Since obesity is the most important predisposing factor in the development of diabetes one would suppose that the prevailing mode for slimness would favourably affect the weight of the female and thus reduce the incidence of diabetes. Apparently, reducing has not affected the female population over fifty years of age.

In Chart 5 an analysis is made of 200 consecutive private cases as to sex and age at onset. There is a rise just before puberty, which is greater in males. The greatest rise occurs in females between 45 and 50; there is a rise in males between 50 and 55 which is not so great as the rise in females. Joslin,[3] in a similar analysis of several thousand cases seen in New England, shows a curve of the same general shape. It is interesting that two curves showing the age-incidence of the onset of diabetes should show such a marked similarity. The one is taken in an old, settled, prosperous part of the country on thousands of cases, the other is taken on a small number of cases in a rural district not long settled. It would seem that the onset of diabetes follows definitely predictable rules according to age and sex, no matter in what environment the study is made. That is, given a hundred consecutive cases of diabetes, a student of the disease could fairly accurately guess the percentages in each decade belonging to either sex.

The age factor.—The variation in rates in different groups is shown in Chart 6. In the under-20 group the rate has not exceeded 3.9 (1919). In the 20- to 49-year group the rate has not exceeded 6.3 (1920), but in the 50- to 59-year group we have a rate of 36 (1931), in the 60- to 69-year group we have a rate of 81 (1933), and in the over-70 group a rate of 170 (1932). Thus we see that the age-groups causing the increase in the rate are not in the under-50 groups. Since 1920 the under-50 groups have shown a downward trend.

In Chart 7 is shown the percentage of total deaths from diabetes in two age-groups. Ever since 1923 the age-group under 50 has contained less than half the deaths. In Chart 8 we see that in Ontario since 1901 the group under 50 had less than half the deaths, again showing that Saskatchewan has repeated Ontario's diabetic history twenty-two years later. Insulin became generally used in 1923; its effects were immediately seen, both in Ontario and in Saskatchewan. In 1932, in Saskatchewan, 81 per cent of the deaths from diabetes occurred in those over 50 years of age.

The race factor.—It has been shown that the incidence of diabetes is high among Jews. Since Jews usually live in cities and there are few cities in Saskatchewan there are not many Jews in the population. Indians are not subject to diabetes. Complete physical examinations, including urinalyses, have been done on 1,500 Indians in Saskatchewan by the Anti-Tuberculosis League, and no case of diabetes has been discovered. Dr. A. B. Simes, who has charge of the medical work in the File Hills Reserve, has not seen a case of diabetes among Indians. One case was reported from the Cowessess band, but this band has an intermixture of white blood. A case was reported from Chesterfield Inlet, of which the details are not known. Mr. W. Murison, Inspector of Indian Affairs in Saskatchewan, has never heard of a case of diabetes in a pure-blooded Indian. The reason is not because they are all thin, for some of the older Indian women are very fat.

SUMMARY

The diabetic death rates in Saskatchewan since it became a province have been analyzed as to age and sex.

Saskatchewan's diabetic death rate is rising.

Because Saskatchewan has a largely rural population and a young average age the rate is low compared to the other provinces, with the exception of Alberta. The rate is low compared to that of England and Wales and to that of the United States.

The death rate from diabetes in the female exceeds that in the male.

The rate is falling in the age-groups under fifty and rising in the age-groups over fifty.

That part of the population most liable to the onset of diabetes is that of the females between 45 and 50.

We can find no evidence to suggest that the Saskatchewan rate will not continue on its upward trend.

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ANTI-PNEUMOCOCCUS SERUM IN PNEUMONIA

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THE use of specific anti-pneumococcus serum was first recorded by Neufeld and Händel[1] in Germany in 1910, and in America three years later by Cole and Dochez.[2] The fact that at first serum was unconcentrated and necessarily used in large amounts made its use a rather formidable procedure. The later development of a method of concentration by Felton[3] (1928) not only made the actual administration simpler but greatly reduced the number and severity of the reactions. In the past few years extensive use has been made of concentrated anti-pneumococcus sera and valuable reports have appeared.

The present communication is based on experience with a group of patients suffering from lobar pneumonia, of whom 89 were found to be either type I or type II. Forty-three of 67 type I cases and 15 of 22 type II cases received treatment with concentrated anti-pneumococcus serum.

While potent anti-pneumococcus serum has been developed for several of the sub-groups of group IV pneumococci, it is particularly in type I or type II pneumonia that specific serum has been found of greatest value. These two types, however, constitute the etiological factor in more than 50 per cent of all cases of lobar pneumonia, as has been shown in many widely separated centres. A report by the Medical Research Council of Great Britain,[4] in 1934, of cases from London, Edinburgh, Glasgow and Aberdeen indicated that 35.5 per cent were type I and 28 per cent type II—a total of 63.5 per cent. We have found the incidence in Toronto somewhat similar (Table I).

In a large group of 9,639 collected from the literature,[5] the type I cases were 33.4 per cent, and type II, 23.3 per cent, *i.e.*, a total of 56.7 per cent.

DETERMINATION OF TYPE

Anti-pneumococcus serum is very definitely type specific. Type I serum does not benefit type II cases, and vice versa. Formerly, when many hours were necessary for the determination of the type, it was often deemed advisable to administer a mixture of sera. That, fortunately, is no longer necessary if sputum is typed by the Neufeld method.[6] Loopfuls of glairy rusty sputum are thoroughly mixed on a glass slide with a couple of loopfuls of undiluted rabbit antiserum of the various types, and two loopfuls of methylene blue. In a mixture containing pneumococci and homologous antiserum the capsule of the organism swells in a manner that plainly differentiates it from the other specimens. This change occurs promptly, so that in the great majority of cases the type may be determined within 30 minutes. When organisms are very few, washed sputum is injected into the peritoneal cavity of a mouse. The exudate is withdrawn in three or four hours and the type determined.[7] When no sputum is available, the throat is swabbed and the swab incubated for four to six hours in glucose broth. In the presence of bacteriæmia the type may, of course, be determined from the organisms recovered from the blood stream. In infants and children pneumococci frequently may be obtained by gastric aspiration. The prognosis, however, in children is so favourable, ordinarily, that serum is rarely indicated.

TABLE I.
DISTRIBUTION OF CASES (PERCENTAGE) ACCORDING TO TYPES

Year	*Cases*	*Type I per-centage*	*Type II per-centage*	*Type III per-centage*	*Group IV per-centage*
1930	56	41.1	19.6	10.7	28.6
1931	62	29.1	3.2	22.6	45.1
1932	69	15.9	7.3	14.5	62.3
1933	46	21.7	6.5	17.4	54.4
1934	46	41.3	6.5	17.4	34.8
1935	25	36.0	16.0	12.0	36.0

REACTIONS

Reactions following the intravenous injection of anti-pneumococcus serum do occur but are infrequent and rarely serious. They are of three types: (1) allergic; (2) thermal; and

(3) delayed. With the last-named we are all familiar, urticaria, arthralgia, and glandular swelling occurring 7 to 10 days after injection, as in the case of other sera.

Thermal reaction is characterized by an early sudden elevation of temperature, lasting an hour or two, associated perhaps with chill, cyanosis and even collapse. When earlier preparations were in use these reactions were frequent and often severe. With the present concentrated serum, however, this is not the case. Of our 58 patients receiving serum, 16, or 27.6 per cent, had chills of some degree, but in only 4, or 7 per cent, was it considered desirable to discontinue treatment before an optimum amount of serum had been administered. None of the serum-treated patients of our series who had a thermal reaction failed to make a satisfactory recovery. The treatment of such reactions is symptomatic. Usually serum administration may be continued cautiously. One should avoid using serum which has a precipitate. Dry sterilized syringes are preferable to those sterilized by boiling, as the presence of water is a factor in the causation of precipitates.

Numerically few, but of more serious significance, are reactions of the allergic type. These may be so mild as almost to escape recognition, or in rare instances may be alarming in suddenness and severity. Allergic reactions may be evidenced by a faint flushing of the skin, a hacking cough, a slight substernal tightness, with or without wheeziness, coming on several minutes after the injection, or by the immediate appearance of giant urticaria, severe asthma, or even collapse and death. Until pneumococcal antibodies can be entirely separated from horse serum the administration of serum involves the remote possibility of death from "anaphylaxis". Such danger is extremely small, and fear of it should not deprive patients of the advantage of specific treatment, subject to proper precaution. In our series we experienced no allergic reactions. In the large group of 956 patients reported this year in a study in Massachusetts,[5] "anaphylaxis" accounted for one death. Nevertheless, it is only by observing such precautions as are detailed below that such a low rate will be assured.

The possibility of allergic reactions from the intravenous injection of this or any other serum may be anticipated in three groups of cases. First, and most dangerous, are the known allergic individuals who have a personal or familial history of asthma, hay fever, etc., particularly when these symptoms are related to proximity with horses. Second, those who have had serum administered previously (unless within ten days). Toxoid contains no serum, but toxin-antitoxin does. Vaccines, of course, do not. Third, a group of unsuspected sensitized individuals revealed only by tests.

The tests commonly used are the ophthalmic and the intradermal. The former consists of the instillation of a drop of serum into the conjunctival sac of one eye. If within 20 to 30 minutes there are itching, lachrymation or marked reddening, as compared with the control eye, the test is positive. For the skin test, one may inject intradermally 0.1 c.c. of a 1/10 dilution of normal horse serum; a control test with saline is advisable. If the test is positive an urticarial wheal will appear within 20 to 30 minutes, possibly with pseudopodia and a pink areola. Very rarely, in the unusually sensitive patient, asthmatic symptoms may result even from this test alone, so that one should be prepared to inject epinephrine—0.5 to 1.0 c.c. of a 1/1,000 dilution.

THE AVOIDANCE OF ALLERGIC REACTIONS

Although no absolute rules can be formulated to fit every individual case, it may be desirable to withhold serum from many patients falling in the above-mentioned groups, including those who give a positive ophthalmic reaction and a strongly positive intradermal reaction, particularly where there is an allergic history, personal or familial. If, however, special precautions are observed serum may be administered with relative safety in other questionable cases where doubt exists as to the advisability of giving serum. These include (1) those who have recently (later than 10 days) received small amounts of serum, (2) or have had large amounts given more than three months ago, (3) those with only a *familial* history of asthma or hay fever, and (4) those with a personal history of only lesser allergic manifestations, such as urticaria, angio-neurotic œdema, eczema, migraine, etc., unless serum sensitivity is the underlying cause.

There is no one method which can be advised for a satisfactory desensitization of all patients;

each case presents an individual problem. The precautionary measures commonly used are similar to those suggested by Besredka for desensitization. First administer epinephrine (0.5 c.c. of a 1/1,000 dilution) and keep a loaded syringe available. After 5 to 10 minutes begin serum administration with 0.25 to 1 c.c. *subcutaneously*. To this dose one may add epinephrine to delay the absorption. If little or no reaction results in 30 minutes, one may give a larger amount, again subcutaneously, repeating at half-hour intervals until 5 to 10 c.c. has been administered subcutaneously. If still no reaction ensues begin intravenous administration with 0.5 to 1 c.c. of serum, slowly given. One may then proceed as with the non-allergic patient.

METHOD OF ADMINISTRATION

With the great majority of patients one may begin treatment with the slow *intravenous* injection of about 6,000 to 10,000 units (3 to 5 c.c.) of serum which has been warmed to body temperature. If a reaction ensues it is usually evident within two hours. Consequently, one waits for that period of time before giving the second dose of about 50,000 units. Two hours later 60,000 units are given, and ordinarily one is able to give another 80,000 units six hours after treatment is begun, thus making a total of 200,000 units. Even a mild reaction makes it imperative that any subsequent dose should be given slowly and reduced in amount. If dyspnœa, collapse, weak pulse, tightness of chest, with or without urticaria, should result, immediate subcutaneous injection of 0.5 to 1 c.c. of epinephrine is indicated. If the reaction has not been severe, and if urticaria has not occurred, the administration of serum may usually be cautiously continued with smaller doses, given slowly. Treatment should be discontinued at once should asthma result, or shock, or severe thermal reaction. As already stated, this in our experience is very rarely necessary.

Some authors recommend that serum be withheld from the old and infirm, the desperately ill, or those in coma. While undoubtedly some of these will die before an effective amount of serum is administered, we have seen patients make a spectacular recovery, due, we believe, to the specific treatment. A case in point is that of a patient "L" who was admitted to Toronto Western Hospital on the fourth day of his pneumonia, with temperature 104.8° F.—later 107.5° F.—pulse 148, and respiration rate of 48. The sputum showed type I pneumococcus. The right middle and lower lobes were consolidated, and subsequently the left lower. His blood culture was positive; he was irrational and for several hours unconscious. In this apparently hopeless case it seemed futile to give specific serum. We did administer it, however, and with a satisfactory result.

Individualization of dosage is desirable, the optimum amount of serum to be administered varying according to such factors as the virulence of the organism, the duration of the disease, and the presence or absence of complications. Type II cases usually require more than type I. The minimum effective amounts are perhaps 120,000 units of type I serum and 200,000 units of type II. Mouse-protection tests, using serum withdrawn from the patient not less than 8 hours after treatment, provided a good index, but are not sufficiently rapid to be of general use. We have had no experience with testing patients' serum for agglutinins, nor with the more promising skin test with type specific polysaccharide as a guide to dosage, but have found the clinical condition of the patient a satisfactory guide. If 12 to 14 hours after 200,000 units of serum have been given obvious improvement in the patient's condition is not evident we usually consider it advisable to continue administration in 60,000 units or 80,000 units amounts up to a total of 400,000 units or 600,000 units, and occasionally more. In the case of the desperately ill patient "L" already referred to we gave 860,000 units over a 4-day period. These larger amounts are indicated also, we believe, if early progress is not maintained, if treatment has been commenced late in the disease, if more than half a lung is involved, if the disease is of severe type, or if the blood culture becomes or continues positive, and when the patient is pregnant or in the puerperium.

Anti-pneumococcus serum is antibacterial, not antitoxic. It does not directly prevent death from cardiac failure or anoxæmia. One should therefore not neglect good general medical and nursing care or the early use of oxygen when indicated.

RESULTS

Our experience leads us to conclude, with Cecil, that there is no more striking effect in the whole domain of specific therapy than that which follows the early administration of anti-pneumococcus serum. Symptomatic improvement commonly begins about 12 hours after the first dose. There is less distress, less laboured breathing, less cyanosis. The patient is clearer mentally and reports he "feels" better. The temperature drops 2 to 4 degrees and usually does not again reach its former height. There may be a secondary rise, however, on the following day. This, if maintained, raises the question of further administration. Usually the temperature, after an initial fall and perhaps a slight rise, quickly trails off to normal. The pulse rate drops more slowly, as does the respiratory rate, though, as stated, subjective dyspnœa is relieved early. As a rule, consolidation of the lung does not extend, but, on the other hand, resolution does not coincide with the drop in temperature, nor does the fall in the leucocyte count. Positive blood cultures frequently become negative within 24 hours.

The Medical Research Council (Armstrong[4]) reports that in serum-treated patients crisis occurred 2½ days earlier in type I, and 2 days earlier in type II cases as compared with controls. In other groups (Edinburgh and Glasgow) 4 to 7 times as many treated type I cases showed defervescence on or before the fifth day, and 3 to 6 times as many type II cases. In our type I cases, 53 per cent and in type II cases, 60 per cent, had a critical drop in temperature within 24 hours of serum administration. It is interesting to note that the remainder, including those whose temperature fell by lysis, received their serum later in the disease.

The effect on mortality is, of course, extremely difficult to assess with any degree of accuracy, owing to the many variable factors. In our small series of 67 type I patients the death rate in the serum treated cases was 23.3 per cent, as compared with 41.7 per cent in the untreated. Of the 10 serum-treated patients who died in only 4 was the pneumonia uncomplicated, and these received serum as late as the six, seventh, eighth and ninth days. Of the remaining 6 one died within 24 hours of admission with delirium tremens, having received only 70,000 units; one had embolic gangrene of foot, and autopsy showed that the lung had cleared; a third died from gastric hæmorrhage. In one case the pneumonia was complicated by miscarriage, in another by pneumococcal pericarditis, and in a third by pneumococcal meningitis. Three of these fatal cases had positive blood cultures, as did 2 of those who recovered. Among the cured patients also was a woman of 63 years who on admission had a blood sugar of 466 mg. per 100 c.c.

Of 15 type II cases who received specific treatment all recovered. Three of these showed pneumococci in their blood stream.

TABLE II.

EFFECT OF ANTI-PNEUMOCOCCUS SERUM ON MORTALITY RATE

Series	*No. of cases*	*Type*	*Special group*	*Mortality rate percentage*	*Mortality rate in controls percentage*	*Relative mortality percentage*
Medical Research Council of Great Britain	140	I	20-40 yrs.	5.7	11.2	51.0
Medical Research Council of Great Britain	111	II	20-40 yrs.	12.6	22.7	58.0
Heffron (collected) ..	2,458	I	not selected	15.6	25.0	62.0
Heffron (collected) ..	670	II	not selected	30.6	41.0	74.6

These numbers are, of course, too few to be of any conclusive value, but when large groups reported on by different individuals from various centres and covering different years are combined considerable accuracy may be presumed. The Therapeutic Trials Committee of the Medical Research Council,[4] having made a careful study of the use of serum in London, Edinburgh, Glasgow and Aberdeen, concluded that in the age group 20 to 40, serum treatment apparently reduced the fatality from type I and type II pneumonia.

In 140 treated type I cases, the death rate was just 51 per cent of the control cases. Of

111 type II cases, the percentage was 55. Lord and Heffron[5] have collected larger numbers which are even more impressive. In 2,458 type I serum-treated patients of all ages the death rate was 15.6 per cent as compared with a corresponding percentage of 25 in 1,614 untreated controls. Similarly, they find in 670 type II serum-treated patients a death rate of 30.6 per cent, as compared with 41 per cent in untreated controls. These figures suggest that of the 757 deaths that occurred last year in Ontario from pneumonia of all types perhaps a third fall into the category of *preventable* deaths.

Both age and pneumococcal bacteriæmia are well recognized as of serious prognostic import. In a small number of patients the Medical Research Council Report found that serum appeared to have little effect on the older age groups or on patients with positive blood culture. In our small series we have 50 per cent mortality in type I untreated cases over 40, while in those receiving serum the death rate was only 26.6 per cent. These figures are supported in reports of larger series. We quote again the Massachusetts Pneumonia Study[5] (Table III). In type I patients, 10 to 49 years of age, the treated cases gave a mortality rate of 8.3 per cent, as compared with 22 per cent in the untreated. At and over the 50 year level there is, of course, a higher fatality rate, but again the treated cases have a great advantage, 30.8 per cent, as compared with 60.7 per cent.

In cases with bacteriæmia, too, the beneficial effect of specific treatment is well illustrated by the figures compiled by Lord and Heffron.[5] In a group of 271 type I cases with positive blood culture the mortality rate was 31 per cent, as compared with 64.6 per cent in those not receiving serum (Table VI). Similarly in type II bacteriæmic cases the mortality was 52.9 per cent as compared with 76.2 per cent.

Thus we see the prognosis is very greatly improved by specific therapy in the later age groups and in cases with bacteriæmia.

The greatly enhanced value of serum given early in pneumonia as compared with later administration is evident from a study of all published figures. Some authors even state that serum is valueless after the fifth day. While this may be an exaggerated view, there is no doubt that the death rate is greatly reduced when specific treatment is given prior to the fourth day. The Massachusetts Pneumonia Study[5] reports a death rate of only 11.1 per cent in 504 type I cases treated within 96 hours, and in 377 cases treated within 72 hours the rate was only 8.5 per cent (control 25 per cent). One hundred and thirty-six type II

TABLE III.

EFFECT OF SERUM IN DIFFERENT AGE-GROUPS

Series	*No. of cases*	*Type*	*Special group*	*Mortality rate percentage*	*Mortality rate in controls percentage*	*Relative mortality percentage*
Massachusetts Pneumonia Study[5]	432	I	10-49 yrs.	8.3	22.0	37.7
Massachusetts Pneumonia Study[5]	65	I	50 yrs. and over	30.8	60.7	50.0

TABLE IV.

THE EFFECT OF SERUM IN PATIENTS WITH AND WITHOUT BACTERIÆMIA

Series	*No. of cases*	*Type*	*Special group*	*Mortality rate percentage*	*Mortality rate in controls percentage*	*Relative mortality percentage*
Heffron (collected)	271	I	positive blood culture	31.0	64.6	49.4
Heffron (collected)	68	II	positive blood culture	52.9	76.2	69.4
Heffron (collected)	191	I	negative blood culture	5.2	26.2	19.8
Heffron (collected)	54	II	negative blood culture	16.7	54.2	30.8

cases treated before 96 hours had a 27.2 per cent mortality, as compared with 41 per cent in the controls.

Pneumonia is, therefore, a medical emergency which, like appendicitis, calls for prompt action on the part of the practitioner. In too many cases specific therapy has been delayed so long that large amounts are required and maximum benefit cannot be expected. This delay is chiefly due to the fact that we have been trained to diagnose pneumonia by certain well-defined physical signs which frequently are of slow development. Is it advisable always to await these passively? A flat x-ray film will often disclose a deep-seated pneumonia.

TABLE V.
THE EFFECT OF EARLY SERUM TREATMENT

Series	*No. of cases*	*Type*	*Special group*	*Mortality rate percentage*	*Mortality rate in controls percentage*	*Relative mortality percentage*
Massachusetts Pneumonia Study	504	I	treated within 96 hrs.	11.1	25.0	44.4
Massachusetts Pneumonia Study	377	I	treated within 72 hrs.	8.5	25.0	34.0
Massachusetts Pneumonia Study	136	II	treated within 96 hrs.	27.2	41.0	66.3

But simpler and more important than anatomical localization is specific diagnosis by early recovery of pneumococci from sputum or blood stream. If type I or II are found, this fact provides strong presumptive evidence of lobar pneumonia, as, unlike III or IV, type I and II organisms are rarely found apart from a patient ill with that disease or those in close contact with him. As shown in Table VI, no type I pneumococci were demonstrated in the throats of 112 healthy persons each examined on three occasions, and type II was found only once. On the contrary the incidence of type III and Group IV was 12.5 and 86.25 respectively. It is thus possible frequently to anticipate physical diagnosis and prepare for specific therapy early in the disease. The advantage of this is clear.

TABLE VI.
THE INCIDENCE OF PNEUMOCOCCI IN THE THROATS OF HEALTHY PERSONS (BROWN)
112 PERSONS TESTED THREE TIMES EACH

Type	*Cases showing pneumococcus*	*Incidence percentage*
I	0	0
II	1	1.25
III	10	12.50
Group IV	69	86.25

SUMMARY

1. About 50 per cent of lobar pneumonia in Toronto is caused by pneumococcus type I or II.

2. The type of pneumococcus in sputum may be quickly determined by a simple method (Neufeld) which does not require elaborate equipment.

3. Results in the group of cases type I and type II reported, as well as in large series reported by others, indicate that serum treatment causes very definite symptomatic improvement, hastens defervescence, and reduces mortality. These improved results are found in all classes, young or old, with or without bacteriæmia.

4. Early specific diagnosis and treatment are of great importance. An expectant attitude can no longer be justified. Lobar pneumonia is a medical emergency.

5. Serum treatment may, with due precaution, be administered by the family physician in the patient's home.

Acknowledgment is made to Dr. C. E. Cooper Cole and Dr. O. J. S. Little for permission to include eight cases treated on their wards at Christie Street Hospital. Of the other cases reported, all but a few private patients were treated on the Medical Wards, Toronto Western Hospital, service of Dr. F. A. Clarkson. Appreciation is also expressed to the Director of the Connaught Laboratories, University of Toronto, for the serum used in this study; also to Miss Elizabeth Anderson for her technical assistance and painstaking work in abstracting histories.

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THE TOXÆMIAS OF PREGNANCY*

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THE toxæmias of pregnancy have been, and still remain, the enigma of obstetrics. Coming often unheralded — indeed, the term eclampsia signifies a *flash* — attacking particularly the young primipara, attended with a high mortality both to mother and babe, the toxæmias have engaged the minds of countless observers, and still the cause remains unknown, and the very name, toxæmia, begs the question.

As to the seriousness of this complication of pregnancy, let the statistics of maternal mortality in the three prairie provinces tell the tale. In the nine years, 1926 to 1934 inclusive, there were in Manitoba 113,526 births and 629 maternal deaths, of which 133, or 21.14 per cent, were due to eclampsia, albuminuria of pregnancy, and pernicious vomiting of pregnancy; in Saskatchewan 188,543 births, with 916 maternal deaths, of which 160, or 17.46 per cent, were due to toxæmias of pregnancy; and in Alberta 146,218 births, and 825 maternal deaths, with 165 deaths, exactly 20 per cent, credited to toxæmic conditions. Thus we had in Manitoba, Saskatchewan and Alberta, provinces which are remarkably similar in physical contour, types of population and social conditions, in the years 1926 to 1934 inclusive, a total of 448,287 births and 2,370 maternal deaths, of which 458, or 19.33 per cent, were due to toxæmias. Nor does the tale of deaths completely sum up the situation, for it takes into account neither the maternal morbidity and the lessening of life expectancy nor the fetal deaths. Of all maternal diseases toxæmia is the most lethal to the unborn child. Eardley Holland in his investigation of 301 fetal deaths found that 77, *i.e.*, 26 per cent, were caused by albuminuria, eclampsia, or accidental hæmorrhage. Moreover, many children born alive by toxæmic mothers succumb shortly after birth.

The theories as to causation of the toxæmias are many. Indeed, there are those who deny that the conditions listed under that name are due to the presence of a circulating toxin. Paramore claims that eclampsia results from changes in intra-abdominal pressure, and G. W. Theobald, that all the ailments and toxæmias associated with pregnancy are caused by deficiency of some substance or substances in the diet, the most important of which is calcium. Stander, in Williams' "Obstetrics", lists the theories under twelve headings:— uræmia; bacterial origin; auto-intoxication; biological reactions; entrance of fetal elements into the maternal circulation; fetal metabolic products; placental decomposition products; alterations in maternal metabolism; endocrine disturbances; mammary toxæmia; effect of dietary alterations; physico-chemical changes.

It is clear that the cause of eclampsia has not yet been discovered, in spite of the enormous amount of research, especially along biochemical, serological and immunological lines. The facts which have to be accounted for by any acceptable theory are as follows:—

1. The toxæmias are associated with the presence of the products of conception in the maternal organism, but may arise without actual development of a fetus, since they may occur in connection with a mole.

2. Removal of the products of conception, or even death of the fetus, usually leads to rapid recovery.

3. The condition is more frequent in primiparæ, in multiple pregnancy, and in hydramnios.

4. Eclampsia is more common in northern countries than in the tropics.

5. Its incidence increases as pregnancy approaches term.

6. Marked œdema is usually a favourable sign, while its absence adds to the gravity of the prognosis.

7. In both eclampsia and pernicious vomiting, terminating fatally, degenerative changes are found in the liver and kidneys. The characteristic liver lesion in pernicious vomiting is a central necrosis of the lobule, while in eclampsia the degenerative process begins in the peri-portal area.

8. In eclampsia, thrombosis, necrosis and hæmorrhage are frequently found.

9. In a very high percentage of cases of toxæmia albuminuria is found, as might be expected from the renal involvement.

10. Hypertension, albuminuria and convulsions are characteristic accompaniments of eclampsia.

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11. Anoxæmia, resulting from deficient oxidative processes, is commonly present.

12. Repeated eclampsia rarely occurs, whereas chronic nephritis gives rise to increasingly serious trouble in each succeeding pregnancy.

13. Eclampsia is not confined to the human race but occurs in mares, cows, ewes, sows and bitches.

PHYSIOLOGICAL CHANGES IN NORMAL PREGNANCY

Even the normal pregnant woman presents certain abnormalities. Her metabolism is affected by the retention of material required for the construction of new fetal and maternal tissue; changes occur in the liver and kidneys; and there are also other abnormalities.

The first half of pregnancy is characterized by the excretion of more nitrogen than is ingested in the food, while in the second half the reverse is the case. During the latter half of pregnancy Nature is a very thrifty housekeeper. The effect of the lowered nitrogen output in this period is seen in the lessened urea content of the blood, usually less than 20 mg. per 100 c.c.—secondly the urea content of the urine is reduced relatively to the other nitrogenous constituents. Considerable storage of inorganic salts also takes place, notably calcium, phosphorus and magnesium. The fat and cholesterin content of the blood is increased during pregnancy, falling to normal with the onset of lactation.

Some observers, working with the lævulose tolerance test, and determining the bilirubin content of the blood and the presence of urobilin in the urine, have thought that the liver of pregnancy showed evidence of biliary stasis, or a mild impairment of function, but their findings have not been generally accepted. It is well known, however, that there is a lowering of the renal threshold for sugar. In pregnancy there is a tendency to hydræmic plethora. The solid content of the serum is diminished, its water content is increased, and there is a moderate rise in blood volume. This may indicate a slight defect in the capacity of the kidney for water excretion.

Even in normal pregnancy a slight degree of acidosis is present, *i.e.*, there is a slight diminution in the alkaline reserve of the blood. The ammonia index of the urine is raised. Acetone bodies are produced more readily in pregnancy. The blood plasma of the pregnant woman contains an excessive amount of fibrinogen. There is a definite elevation in the basal metabolic rate in the latter half of pregnancy. It appears, therefore, that in normal pregnancy there are departures from the standard physiology of the non-gravid, and that some of these abnormalities in exaggerated form are found in the toxæmias.

CLASSIFICATION OF THE TOXÆMIAS

The toxæmias may be classified as follows:—(1) pernicious vomiting of pregnancy, (hyperemesis gravidarum); (2) albuminuria of pregnancy (the low-reserve kidney of Stander); (3) pre-eclampsia; (4) eclampsia; (5) chronic nephritic toxæmia. In addition there are other conditions which may be grouped under the toxæmias: excessive salivation or ptyalism, acute yellow atrophy of the liver, polyneuritis and dermatitis herpetiformis. Salivation is usually associated with excessive vomiting, and acute yellow atrophy may be caused by agents which have no connection with pregnancy. Pernicious vomiting is usually confined to the first three months of pregnancy, and the others to the last three months, but there are numerous exceptions. The albuminuria of pregnancy (low-reserve kidney) is characterized by the presence of albumin in the urine, but symptoms are nil or slight, the blood pressure is not raised, œdema is not marked, and the prognosis is good. Pre-eclampsia and eclampsia are associated with increased blood pressure, usually over 140 systolic and reaching 200 or even higher, large amounts of albumin in the urine, œdema of varying degree, and sometimes retinal changes. With eclampsia there are usually one or more convulsions followed by coma. With recovery, the blood pressure as a rule returns rapidly to its former figure.

Strictly-speaking, chronic nephritic toxæmia should not be classed among the toxæmias, as it is really a pre-existing nephritis complicated with pregnancy. However, it is often difficult to make a differential diagnosis between this condition and pre-eclampsia, and the existence of an antecedent nephritis is not always known. We must admit that the renal function tests we possess at present are not wholly satisfactory, and that kidneys which stand up well under

the strain of everyday life break down under the added load of pregnancy. Pregnancy is the most delicate test of renal function.

THE PATHOLOGY OF THE TOXÆMIAS

Fatal cases of pernicious vomiting show diffuse fatty changes in the liver, and in some cases an area of central necrosis in the liver lobule similar to that seen in chloroform or phosphorus poisoning.

In eclampsia the characteristic changes in general are those due to thrombosis, hæmorrhage and necrosis. The liver shows degeneration and necrosis in the periphery of the lobule and hæmorrhage either under the capsule or throughout the substance of the liver. The kidneys show alterations in the vessels and epithelium of the glomeruli, together with degenerative changes in the convoluted tubules. The brain shows congestion, œdema, hæmorrhage and thrombosis. Hæmorrhage and necrosis are also found in the heart, lungs, suprarenals and pancreas.

PROPHYLAXIS OF THE TOXÆMIAS

Stander states that about 10 per cent of pregnant patients suffer from one or another of the toxæmias of pregnancy. Now, no matter what views are held as to the etiology of the toxæmias, it is generally agreed that their incidence can be reduced, or at any rate the graver manifestations can be prevented in the majority of cases. This can be done only by adequate prenatal care. This involves not only a general physical examination of the patient, the elimination of focal infections, and systematic and repeated examinations of the urine, blood pressure, body weight, and blood if indicated, but also an appraisal of the mental attitude of the patient to her pregnancy and labour, and of her economic position. Much toxæmia would be eliminated if women would consult their family practitioner or an obstetrician before pregnancy, rather than after. In other words the patient should find out whether she is fit to conceive.

At the first consultation a careful history should be taken. One should ask particularly for a history of scarlet fever, diphtheria, nephritis, rheumatism or tonsillitis. The menstrual and obstetrical history should be investigated and recorded. At the first examination of the urine the reaction, specific gravity, presence or absence of albumin and sugar, and, if indicated, a microscopical examination of a centrifuged specimen for pus, blood, or casts, should be recorded. Infections of the urinary tract are not uncommon and contribute to toxæmia. At subsequent examinations the urine should be tested for albumin and the specific gravity noted. Patients should be instructed to note any marked reduction in the amount of urine passed. The blood pressure, both systolic and diastolic, should be recorded at each examination. Any reading over 140 systolic should call for close scrutiny of the patient. It is important to note the patient's weight at each visit, as failure to gain means under-nourishment, while a sudden rise in weight may be the first indication of disturbance of water secretion. Constipation should be avoided by advice as to food, exercise, and the drinking of large quantities of water. If necessary, mild laxatives such as liquid petrolatum, milk of magnesia, or compound liquorice powder may be ordered. Vomiting calls for hospital treatment if it is not kept within bounds when the patient takes "little but often" of proper foods, has sufficient sleep, rest and fresh air, and when a bromide mixture is exhibited. Delay may be costly and dangerous. LaVake states that the following signs call for therapeutic abortion in pernicious vomiting: a pulse rate rising above 120 and not reducible by rest, hydration and glucose administration, mental wandering or delirium and stupor, and signs of toxic myelitis.

Regarding diet, we have learned much from the physiologist and biochemist. Every pregnancy is a gigantic biochemical experiment in which a new organism is created, nurtured, and sheltered for nine months, then more or less precipitately expelled, and thereafter ideally nourished again from the breast for nine months more. For the building of fetal tissues, protein, carbohydrates and mineral salts are essential. In the first three months of pregnancy the carbohydrates are especially necessary to prevent depletion of the maternal stores of glycogen in the liver and to maintain its deaminising power. Lack of carbohydrates may cause an upset in carbohydrate metabolism with resultant vomiting. In the middle three months of pregnancy the proteins are indicated, and in the latter half the minerals are especially indicated. It is wise, however, to restrict the amount of meat taken in the last two months of pregnancy, as its excretion may throw too great a strain on the kidneys. It is well known that if the mother's

diet does not supply the needs of the fetus it will draw on the mother's tissues. An experiment on a huge scale took place in Germany during the war years, 1917, 1918, 1919 when lack of proteins and fats led almost to starvation of the population. The children born during that period were slightly heavier than control children born in the period 1910-1912, and a striking decrease occurred in the incidence of eclampsia. Just before the Great War its incidence was about 2.0 per 1,000 births, while in 1918 it was 0.6, *i.e.*, less than one-third, but in 1922 there was an increase up to 2.2 per 1,000 births.

The minerals required are calcium, phosphorus, iron and iodine. Calcium and phosphorus are necessary for the formation of the fetal skeleton. They are found in milk, green vegetables and egg yolk. If calcium is not supplied in the diet it will be withdrawn from the mother's skeleton. In extreme deficiency the mother may suffer from osteomalacia. Calcium is also sedative to the nervous system and so tends to prevent mental irritability. Nutritional anæmia is not uncommon in pregnant women and may contribute to toxæmia by reducing resistance. It is best combated by iron-rich foods, meat, fresh green vegetables, eggs and whole cereals. Kemp, of Vancouver, believes that deficiency of iodine may result in idiopathic stillbirths, *i.e.*, those for which no evident cause of death can be found. Iodine is furnished by sea-foods, such as salmon, haddock, halibut, oysters, and also by iodized salt. Harding and VanWyck, of Toronto, have pointed out the dangers of ingestion of large quantities of table salt, since excessive chlorides throw a great strain on the kidneys and lead to retention of water. Certainly any patient with a tendency to water retention should be instructed to restrict the use of salt.

Vitamins are essential to growth and well-being. They are supplied by citrus fruits, milk, egg-yolk, yeast, whole cereals, cod liver oil, sunlight, wheat germ, carrots, nuts, and green vegetables. Vitamin B is indicated when there is polyneuritis. Exercise in the open air, sufficient sleep, and freedom from worry are powerful factors in the prevention of toxæmia. The doctor should make every effort to keep the patient's mental condition stable.

THE TREATMENT OF ESTABLISHED TOXÆMIAS

All but the very mild forms call for rest in bed, preferably in a hospital. Good nursing is important, especially in pernicious vomiting. In treating this condition all visitors should be excluded. The other principles of treatment of hyperemesis have been mentioned earlier.

In pre-eclamptic toxæmia medical treatment should not be persisted in for more than a few days if there is no improvement. It is better in such cases to induce labour. In eclampsia the best treatment is that of Stroganoff or one of its modifications. Accouchement forcé and Cæsarean section have no place in the treatment of eclampsia, *i.e.*, after convulsions have occurred or when the patient is in coma. Treat the symptoms and assist delivery only when the usual indications for intervention are present. In other words when eclampsia supervenes be a physician first, then an obstetrician.

Chronic nephritic toxæmia causes intrauterine death of the fetus more frequently than does pre-eclampsia or eclampsia, therefore, earlier interference in induction of labour is desirable. Indeed, therapeutic abortion in the first two months of pregnancy is definitely indicated when the mother has had a bad obstetrical history in this connection.

Finally, toxæmic patients who recover should, if possible, be kept under observation for months or even years until the presence or absence of damage to kidneys, liver or heart can be ascertained.

SUMMARY

The toxæmias of pregnancy constitute a serious menace to the well-being, health, and even life of the expectant mother, and to her babe. The cause of eclampsia is not known. With proper care, and especially proper prophylaxis, the incidence and severity of the toxæmias can be greatly reduced.

A COMPARISON OF TOTAL HYSTERECTOMY VERSUS SUPRA-VAGINAL HYSTERECTOMY*

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IN March, 1930, for the Osler Lecture before the Vancouver Medical Association, I reviewed every hysterectomy and myomectomy that I had performed since coming to Vancouver in 1913, reporting all morbid cases in detail. Now, I want to bring the list to date, comparing the results of sub-total and total operations, and giving in detail a technique that has been found helpful.

Our routine surgical treatment is as follows. As soon as the patient is wheeled into the operating room she is put in stirrups, in the lithotomy position, with the hips at the end of the table, and vulvar and vaginal preparation is done with equal parts of tincture of iodine and alcohol on swabs held by an artery or dressing forceps, paying attention to the folds in the vagina and vulvæ. The bladder is catheterized with a soft rubber catheter and a bimanual examination is done and the pre-operative diagnosis confirmed or disproved. A weighted vaginal speculum or, if not feasible, a small Sim's speculum, is placed in position and the cervix drawn down for inspection. The cervical canal is iodized with a cotton applicator. If a diagnostic curettage be indicated it is done, and the pathologist's opinion immediately obtained. If a biopsy be indicated and has not previously been done, a section is taken with the scalpel, being careful to include in the section both normal and abnormal tissue if possible. The cut area is immediately cauterized, thereby avoiding any risk of dissemination if cancer be present. If it be decided to operate from below, the operation is then done. If the operation is to be done from above, the patient is released from the lithotomy position and prepared for the abdominal operation. The surgeon re-scrubs, and is gowned and re-gloved. We wish to urge in all cases, even if it has been definitely decided to operate from above, that the surgeon do this preliminary examination under the anæsthetic. It fortifies or disproves the pre-operative diagnosis, it empties the bladder, and it surgically prepares the vagina and lower cervix which are always contaminated. It takes but a few minutes, and the surgeon does not need to be gowned or the patient draped.

If a vaginal hysterectomy be indicated the routine technique is as follows. The cervix is drawn down. The anterior vaginal wall is grasped with thumb forceps about midway from urethra to cervix, in the median line, and the vagina cut through with scissors, blunt-pointed and curved on the flat, which are then insinuated, first, above towards the urethral meatus and the anterior vaginal wall incised, and, then, backwards to the cervix, extending this incision which now circumscribes the cervix. The vagina is stripped off the bladder and the bladder off the uterus. The peritoneum in the anterior cul-de-sac is opened and an exploration of the fundus uteri is made with the finger. The fundus is then drawn through this anterior peritoneal opening by successive bites with towel forceps curved on the flat. The round ligaments are separately tied and clamped and cut on both sides, then the rest of the broad ligament on the one side and then on the other side, the uterus being removed. If one wishes, long straight Ochsner forceps can be applied on each broad ligament from above and from below, close to the uterus, after first opening the posterior cul-de-sac, the so-called Mayo technique. The round ligaments are first sutured together, the peritoneal cavity closed and the broad ligaments tied and sutured together. Whatever plastic work on the vagina is indicated is then done. The sutured round and broad ligaments are usually sutured to the sub-pubic tissues, especially if it is felt wise to support the bladder. The vaginal incision, through fascia and mucosa, is closed with mattress sutures of 40-day chromic gut No. 2,

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placed far out towards the lateral vaginal walls, the excess of tissue removed, and interrupted catgut closes the vaginal incision. Drainage is practically never used. If only a vaginal myomectomy be indicated, and that myoma be present on the fundus, the anterior vaginal wall is alone incised, without circumscribing the vagina around the cervix. If the myoma be intra-uterine the anterior cervical wall may require incising longitudinally in the centre line.

The routine abdominal operation on the uterus is as follows. The patient is placed in a fairly high Trendelenburg position. The surgeon stands on the patient's left, and the incision is made from the pubes to the navel, or above, a half to three-quarters of an inch to the left of the median line. Bleeding points are caught and tied with No. 0 plain gut, taking care not to grasp more tissue in the forceps than is necessary. Towels are clipped around the skin edges. The anterior sheath of the left rectus is incised well down to the pubes, taking care not to injure the pyramidalis. The inner edge of the rectus sheath is stripped off the rectus to the linea alba and the vessels from the muscle to the sheath are clamped, cut and tied. The rectus is then drawn over to the left, exposing the posterior rectus sheath and peritoneum. We try to avoid grasping the anterior fascia with artery forceps, both on entering and leaving the abdomen, as, wherever a clamp is placed a potential necrotic area is produced, inviting infection. The posterior sheath and peritoneum are carefully opened. At times the upper ends of the deep epigastric vessels require clamping and ligaturing. As soon as the peritoneum is opened, if the patient be well anæsthetized and in good position, air rushes into the cavity carrying the intestines toward the upper abdomen. The gall bladder, liver, foramen of Winslow, pylorus, kidneys and appendix are quickly examined. The pelvis is carefully palpated, and the uterus delivered or grasped with the surgeon's left hand and held up and forward while the tubes and ovaries are visualized. The bladder is orientated as well as the sigmoid and rectum. If a pack be required, care should be taken that it is not too hot, that it is moist, and the omentum should be pulled down over the intestines, placing the pack on the omentum, thereby traumatizing the intestine less. Self-retaining retractors are never used. Good anæsthesia, a resourceful assistant, a long incision, the Trendelenburg position, all help to render superfluous the need of self-retaining retractors which must injure the tissues. Gentleness and strategy overcome many difficulties in the surgery of the abdomen and render the patient more comfortable later. All adhesions must first be separated, keeping close to the uterus and working down and out. A proper orientation is ever necessary.

If a myomectomy be indicated place the uterine incision so that there will be something to peritonize it. In the anterior wall the bladder can be mobilized easily and used, or, if necessary, the round ligaments can be stretched to cover the incision. In the posterior wall it is a little more difficult to peritonize, but the sigmoid can often be used to advantage. In doing a myomectomy the incision is deepened to the myoma and enucleation done, endeavouring to tie the feeding vessels and to approximate the walls with buried catgut, chromic No. 1, only tying tight enough to close all dead spaces. If the endometrium be opened it should first be closed. A buttonhole suture of chromic catgut No. 1 closes the superficial uterine incision, with, at times, the sacrifice of some of the peritoneal flap, and a running suture peritonizes all. It is surprising what large myomata can be removed, preserving the uterus, to the patient's great advantage. If the myoma be in the broad ligament, enucleation can first be started by splitting the broad ligament between the round ligament and the Fallopian tube. If the myoma be low down the bladder should be mobilized early, to prevent injury. In doing uterine surgery the position of bladder and ureters must constantly be kept in mind, and, if there be doubt, the ureters must be demonstrated. For years, we have given the patient methylene blue, gr. 5 by mouth the day before the operation, as a guide in case of injury to bladder or ureters. It is a continuation of a suggestion of Crile who used it as a preliminary to hernia operations. On two occasions, both operations for cancer of the fundus which had eaten through the anterior wall and fixed the bladder, after separating the bladder from the uterus a small trickle of blue was noticed where the bladder

had lost the small area that was adherent. Immediate suture was done with primary union. The methylene blue prevented a fistula. Both patients are still alive more than five years after operation.

The routine technique for a total hysterectomy and the removal of both tubes and ovaries is as follows. The surgeon elevates the uterus with his left hand, taking for granted that all adhesions have first been separated, and the assistant retracts the right abdominal wall. The surgeon elevates, or puts on stretch, the right infundibular pelvic ligament, and, with No. 1 chromic gut suture on a Kelly needle, he first catches the peritoneal edge of this ligament and then transfixes the broad ligament below the ovarian vessels, finding a clear space to pass the needle. The vessels are tied with the ligament lax. The ovarian vessels are picked up with an artery forceps and again tied with No. 0 plain gut. The right round ligament is next elevated with thumb forceps and a Kelly needle, threaded with No. 1 chromic gut, passed below it, again in a clear space, and tied. A hysterectomy clamp is next placed at the right uterine cornua, extending down to the clear space below the appendages, and the round ligament and the broad ligament are cut through. If there be a large tumour and the infundibular ligament difficult to put on stretch, or the vessels difficult to visualize, a finger is shoved through underneath the ovarian vessels and forced through the anterior part of the broad ligament, first above and then below the round ligament, and the vessels and round ligament, separately, are clamped, cut and tied.

We endeavour, if possible, never to transfix a vessel with a needle, thinking that thereby we lessen the risk of embolism and phlebitis and make our field of operation easier to work in. Ligatures are left long on the round ligament and usually on the ovarian vessels. The anterior reflection of peritoneum above the bladder on to the uterus is cut across and the left appendages similarly dealt with. The bladder is stripped down well on to the vagina. At times it is necessary to clip a few fibres in the centre line. One cannot separate this anterior flap too superficially, as, fortunately, the bladder always shoves off with this peritoneal reflection. This separation is extended far enough out on each side to carry the ureters out of harm. The loose tissue along the side of the uterus is clipped down to the uterine vessels, which are always seen and felt before tying or clamping. The posterior reflection is then, less easily, stripped down. If the hysterectomy be done for carcinoma, the uterine vessels are tied well outside, but with, first, a view of the course of the ureter. Otherwise, the uterine vessels are clamped in two places and cut between, bearing in mind the blood supply of this lower lateral uterine area. The fascia surrounding the cervix is then carefully cut through with the scalpel, using the two, previously placed, cornual uterine clamps to elevate the uterus, and the latter is gradually shelled out until the vagina appears. A clamp is required to stop oozing on the anterior lateral area, on both sides, and also on the utero-sacral ligaments. A protective pad is placed in the posterior cul-de-sac. The vagina is opened and the cervix circumscribed, placing Ochsner clamps on the opened vaginal wall, in front and behind, in order to prevent any spill from the vagina, which is often full of blood that has been squeezed out of the uterus in the previous manipulations. At times, it is necessary to place one or two forceps on bleeding vaginal vessels. The vagina is closed with a buttonhole suture of 40-day chromic gut No. 2 on a Mayo needle. The protective pad, placed to receive the vaginal spill, is removed. The lateral clamped lower uterine vessels are doubly tied with No. 1 10-day chromic gut, and the base of the broad ligament is closed with a mattress suture that takes the utero-sacral ligament and edge of vagina in its bite. The round ligaments are sutured into the stump of the vagina with a 40-day chromic No. 2 gut on a Mayo needle, after first being sure that all oozing has been completely controlled, and the vaginal stump and opened broad ligaments are completely peritonized by means of a purse-string suture of No. 1 10-day chromic gut on a Kelly needle. All pads are accounted for, any clots removed, the sigmoid is tucked back into the cul-de-sac, the omentum drawn down, and the abdominal wall closed.

If a supra-vaginal hysterectomy only be done the endometrium lining the cervical canal may be removed. The stump of the cervix should be mattressed carefully in order to prevent opening later when the catgut is

absorbed, with possible post-operative hæmorrhage, about the tenth day. We now use 40-day chromic gut No. 2. We always save the ovaries and tubes, if healthy and a good blood supply can be maintained to the ovary, no matter what the age of the patient, unless the uterus be malignant or questionably so. We practically never drain through the vagina, always closing it snugly, but being careful to see that no oozing is left to produce a hæmatoma. In closing the abdominal wall we endeavour to keep the peritoneal edges rolled out, using a running suture of 10-day chromic gut No. 1 coming back uniting the muscle to the linea alba and tying at the starting point. A doubled 10-day chromic gut No. 1 closes the fascia, a No. 0 plain gut the superficial fascia, and skin clips the skin. We never use stay sutures. We endeavour to deal gently with all tissues, avoiding clamps on muscle or fascia, knowing that devitalized tissue is a potential infected tissue, avoiding too tight sutures, making rigid hæmostasis, etc. During the operation the anæsthetist gives sub-mammary saline. Post-operatively, if necessary, we give an intravenous injection of 5 per cent glucose in normal saline and, if necessary, a blood transfusion. In doubtful cases, where the patient has lost much blood before coming into hospital, she is transfused before operation, or, grouped and a donor chosen, ready for a transfusion as soon as the operation is over. The appendix is usually removed, especially if the patient be a young woman, has had an attack, or where the calibre of the appendix is narrower at the cæcal end. Only occasionally now do we remove the gall bladder for infection or cholelithiasis while doing a hysterectomy. It is done ten or twelve days later, or later still if thought wise.

We are inclining more and more towards total hysterectomy, reserving supra-vaginal hysterectomy for myomata cases in nulliparous women. The total operation is almost as easily performed if care be taken to visualize the blood supply of the lower segment. It is less dangerous, in that in doing the supra-vaginal operation the infected cervix has to be cut through, contaminating the field. There is more tendency to post-operative bleeding where the cervix is left than when it is removed. The patient is saved the risk of later cervical cancer. There is no post-operative cervical or endocervical discharge to worry the patient. She avoids the discomfort of the protruding cervix which occasionally occurs in the partial operation in women inclined to prolapse, even if the round ligaments have been sutured into the stump during the operation. There is no more risk of injury to the ureters in the total operation. In fact, we believe there is less risk, as in the total operation the bladder is stripped off more thoroughly, thereby carrying the ureters farther out and away from danger. There has been no ureteral injury in our series. Occasionally, where a hysterectomy has to be done in advanced cases of endometriomata of the ovaries, with attendant drawing up of the posterior cul-de-sac and union of the rectum to the uterus, it is wise to desist from the total operation for fear of injury to the rectum.

Our post-operative care is very simple. Morphia is ordered p.r.n. for two or three days. The Trendelenburg position is maintained for twelve hours. The catheter is used only if necessary but often enough to avoid over-distension, and, if it has to be used, it is continued after the patient voluntarily voids, once daily, immediately after voiding, until the residual urine is less than half an ounce. An enema is given, if necessary, on the third to the fifth day and every second day thereafter. The clips are removed on the seventh day and the wound overstrapped. A head-rest is given on the 8th to 10th day and the patient allowed out of bed, usually, on the 12th day. Following a suggestion of Walters, for some years we have been using thyroid extract, beginning the 3rd day and continuing until the patient leaves hospital, as a preventive of embolism. The patient is examined in the office one month after operation, and later, as often as necessary. Cancer cases are watched carefully for years. At the office examination, usually about four weeks after operation, we often find a small granulation at the vaginal scar in the vault of the vagina. It is removed with a small cautery. If exposure be difficult, the knee-chest posture is used.

We have a complete record of histories of all patients operated on, with the operative findings, pathological findings and post-operative and present condition. This has not been difficult, as practically all our patients have been

private or referred cases. For the Osler lecture in 1930 all the morbid cases were discussed in detail. Since then there have been three operative deaths: one from coronary sclerosis with angina ten days following a supra-vaginal hysterectomy for myomata in a woman, aged 48, in whom x-ray treatments had failed to check excessive bleeding; one from pulmonary embolism 14 days after a total hysterectomy and bilateral salpingo-oöphorectomy in a woman aged 54, for a chronic infective uterus with a bleeding cervix that the pathologist reported suspicious or pre-cancerous; and one following an emergency supra-vaginal operation for a ruptured uterus in a woman, aged 37, moribund on admission to hospital, three days after labour began and probably six hours after rupture, with a large baby, eleven and a half pounds and the placenta free in the abdominal cavity; she died the same day. There was one ventral hernia in the series up to the date of the Osler lecture which was repaired at a later cholecystectomy for gall-stones discovered during the hysterectomy, and one ventral hernia since in an asthmatic operated on for cancer of the fundus uteri. She has not yet been repaired.

We have classified the pathological conditions that necessitated the operations of hysterectomy and myomectomy under the following heads: myomata, including adenomyomata and with complications; chronic infective uteri, which included cases where curettings or biopsy were suspicious or cases associated with chronic infected appendages, endometriomata, infected cervices, etc.; cancer of fundus uteri, including two cases of deciduoma; cancer of cervix uteri; fibrosis uteri; complete inversion of the uterus; purpura; cancer of the ovaries; ruptured cornual pregnancy; placenta accreta; ruptured uterus, full term, moribund; complete uterine prolapse.

Myomata.—Hysterectomy was done in 347 cases for myomata uteri. Since the Osler lecture in 1930 the total operation was done in 95 cases of myomata without mortality and the supra-vaginal operation in 77 cases with a mortality of one, already reported in this paper. The vast majority of myomata uteri require no treatment. To urge surgery for a symptomless myoma is reprehensible. That a small myomatous uterus is more likely to become malignant than a normal uterus, we very much doubt. In this series there were 3 cases of sarcomatous degeneration. We have had one case of sarcoma of the cervix uteri, but in that case a myoma was not present. The fear of carcinoma starting in the remaining cervix, where a supra-vaginal amputation has been done, we believe is real. For years, in doing the supra-vaginal operation, we coned out the cervix thoroughly, mattressing the stump carefully. A few post-operative hæmorrhages about the tenth day, when the catgut softened and the cervical ring sprang apart requiring a suture of the cervix per vaginam have modified our technique. At present, if the cervix be not absolutely normal, we do a total hysterectomy wherever feasible and, if not, we cauterize the cervix thoroughly either before, at, or after the operation. If the cervix be coned out, a more resisting catgut should be used.

With the present safety and thoroughness of surgery we believe that radiological measures should be placed on the defensive. To radiate young women and thereby destroy the gonads is no better than the old treatment, of thirty years ago, of bilateral oöphorectomy. In young women with symptom-producing myomata a hysterectomy is seldom necessary. A myomectomy is usually possible.

In this series we found very few uncomplicated myomata. The common complications were old salpingitis and oöphoritis, endometriomata of the ovaries, papillary cysts of the ovaries, three cases with associated cancer of the ovaries, infected cervices, tuberculous salpingitis, parovarian cysts, prolapsus uteri, leukoplakia vulvæ, Meckel's diverticulum, red degeneration of a myoma with occasional necrotic areas in the centre of an old degenerated tumour, carcinoma of the cervix and fundus, once an acute appendicitis, a cancer of the breast, and, fairly frequently, gall stones. Myomata are often associated with pregnancy, but seldom act as a complication. If the myomata be low down in the early months nature usually carries them away from the lower segment as pregnancy advances. In three women, during pregnancy in a myomatous uterus, acute red degeneration required myomectomy which was safely done without sacrificing the fetus. In three cases an inguinal hernia was repaired from the inside by withdrawing the sac and purse-stringing the opening. Cholelithiasis is a frequent accompaniment of myomata uteri. Occasionally a

cholecystectomy is done at the time, but usually is reserved for a later operation. It is a common finding, when palpating the gall bladder during a pelvic operation, to find it very tense, so tense at times that it feels as if a little more pressure might cause rupture. Often these gall bladders are free of adhesions with the foramen of Winslow open. Are they all pathological? We do not think so, as many of these women, when carefully quizzed, have no symptoms. We have occasionally been impressed with the disparity in size of the two kidneys, as if one kidney did most of the kidney function.

We have not noticed any marked difference in the post-operative subjective, menopausal symptoms in women of forty or over when the ovaries have been left or removed. In younger women, where hysterectomy is necessary, there is no question that the ovaries should be left if the case be not malignant. If the Fallopian tubes have to be removed we are careful to preserve the blood supply to the ovaries by making the amputation close to the tube. During this same period of 23 years 129 myomectomies were performed, excluding small myomata removed in course of operations for other pathological conditions without mortality or morbidity. The incidence of pregnancy following in these cases, is small, ten women, but many of the operations were on unmarried women or women in or near the forties where there was a prejudice against losing the uterus entirely. A few were done in older women in association with operations for vaginal prolapse where we wished to save at least some of the uterine body for a support for the bladder. In five of the cases in which myomectomy was done hysterectomy has since been required for recurrence with bleeding.

Hysterectomy was done in 166 cases for chronic infective uteri, which included cases where curettings or biopsy were suspicious and cases associated with chronically infected cervices, chronic disease of the appendages, endometriomata, etc. There were three deaths in this series.

Hysterectomy was done for carcinoma of the fundus uteri in 36 patients with no operative mortality. Four later died of a recurrence, and we think the remainder are still alive, although there are two whom we have been unable to trace.

In doing a hysterectomy for carcinoma of the fundus, and, for that matter, in all uterine surgery, care should be taken not to place a tenaculum in the uterine wall. It also goes without argument that in all radical uterine cancer surgery the liver must first be proved to be free from metastasis. At the vaginal preparation a mattress suture closes the cervix.

Two cases are rare enough to report in detail.

CASE 1

Mrs. S.N., aged 37; operation on March 21, 1927, a case of chorioma malignum. She had one child in January, 1923. Her menses had been normal and were regular on December 26 to 30, 1926. There was a stain on January 2nd and a few clots, and a stain followed for three weeks, after that, no staining. She presented herself at the office saying that she had a recurrence of the cystocele for which we had operated some years before. Examination showed apparently a sub-urethral cyst, the size of a Japanese orange, prolapsed out of the vagina. The uterus was forward and enlarged to the size of a two months' pregnancy and felt so. On March 11th the anterior vaginal wall was incised and an apparent hæmatoma was enucleated, in which were three cyst-like bodies, each the size of a small grape. Dr. Allan McNair reported chorioma malignum. On March 21st, ten days later, a total hysterectomy was done. The uterus was filled with a mass of chorionic cysts. The pathological report was hydadidiform mole which had become malignant, and the microscopic appearance of the tumour was duplicated in the small hæmorrhagic tumour removed from the vaginal wall, which could be safely assumed to be a metastatic tumour. She is now well. The teaching that a molar pregnancy is of great potential malignancy is not borne out by our cases. Recently, the Ascheim-Zondek test is of inestimable diagnostic value.

CASE 2

Miss A.M., aged 48; June 19, 1931. On May 14, 1928, the patient had a diagnostic curettage for menorrhagia with a pathological report of adenomatous polyp and hyperplastic endometritis. She reported again in June, 1931, that almost constant bleeding had occurred since the previous October. On examination the uterus was enlarged and nodular. There was a small nodule in the lower posterior vaginal wall. At operation the small nodule was removed, with immediate examination. Dr. Harry Pitts reported adeno-carcinoma. I felt it would be hopeless to go ahead and remove the uterus, but he suggested it might be the only metastasis and recommended radical operation. It was done. This was one of the two cases previously mentioned where the carcinoma had eaten through the anterior wall of the fundus and invaded the bladder. The removed uterus showed extensive adeno-carcinoma. Some weeks later a small nodule near the urinary meatus was removed with the electro-cautery in the office. There has been no further recurrence.

Hysterectomy was done in 28 cases for carcinoma of the cervix uteri. The gynæcologist meets the terrible scourge of cancer only too frequently. For some reason or other we seldom see cancer of the cervix uteri in its early stages. There is nothing more depressing in our work than to have an otherwise healthy woman present herself with a history of irregular vaginal bleeding and on

bi-manual examination to feel the characteristic irregular nodules which are so hard to describe but so typical when felt. In the early cancer of the cervix, although seldom seen, we believe that radical surgery will give better results than radiological measures. In the early nodular inverting type we incline to surgery, whereas, in the everting cauliflower type we incline to radium. We are fully aware of the excellent statistics reported by some radium clinics, and we will all agree that radical surgery is absolutely out of place in the vast majority of cases when first seen. However, in the early cases that often require a biopsy for diagnosis we believe that cauterizing the cervix thoroughly, followed by total hysterectomy and cauterization of the vaginal incision from above, will protect the patient better than radiological measures. In the borderline case or the advanced case radium at times seems to be life-saving. If a radical operation has been done inspection of the vault of the vagina should be done for years. If a granulation shows, it should be cauterized or radium placed against it.

Hysterectomy was done in only 4 cases of so-called fibrosis uteri, as these cases, uncomplicated, are more safely treated and with less distress by radium, which is 99 per cent efficient.

Hysterectomy was done in 2 cases of inversion of the uterus.

CASE 1

Mrs. E.R.G., aged 37; operation on November 11, 1928, a supra-vaginal hysterectomy being done, saving both tubes and ovaries. Delivery took place at 11 a.m. on November 5th. Labour was slow, associated with inertia. Marked post-partum hæmorrhage followed and the vagina was packed and repacked. On the morning of operation, when we first saw the patient, there was a complete inversion of the uterus. Hæmoglobin was 42 per cent; red cells 2,000,000; blood pressure 70/40. At operation, from above, after vaginal preparation and after ineffectual attempts at reduction from below a unique picture presented itself. The tubes and ovaries were inverted as well into the uterine funnel. The uterus was re-inverted by using the round ligaments as tractors, and, after one of these tore off, the cornua of the uterus were caught with tenacula and gradually reduced. The uterus was markedly congested and the peritoneum was cut and split in many places, potent sources for intestinal adhesions and obstruction, even if the disabled uterus did not develop infection. So a supra-vaginal hysterectomy was done, suturing the stump of the cervix and covering all raw areas. Blood transfusion was done and the patient recovered without incident.

CASE 2

Mrs. J.C., aged 22; operated on March 9, 1930. This patient had had two full-term deliveries, the last one, on December 5, 1929. She had a short labour of eight hours' duration. The placenta was Credéd out inside of half an hour. No manual examination was made, although she reported there was much bleeding and much manipulation of the uterus by massaging the uterus through the abdomen. She was sent to the city at the end of December with the report of a previous curettage and three transfusions of blood. When we saw her on March 9, 1930, her hæmoglobin was 33 per cent; 2,200,000 red cells. The uterus was a sloughing mass, completely inverted into the vagina. At operation it was impossible to re-invert it and a supra-vaginal hysterectomy was done. She recovered without complication. There was a congenital abnormality in that the right round ligament and right Fallopian tube were not attached to the uterus but to the top of the right broad ligament, two inches away from the cornu of the uterus.

The case below, designated as purpura, should not have been operated on.

Mrs. D.A.B., aged 41; operation on July 20, 1922, under local anæsthesia, a supra-vaginal hysterectomy being done. She had a hæmoglobin of 18 per cent, 637,000 red cells, 2,000 leucocytes, with 20 per cent polymorphonuclears and 80 per cent lymphocytes, even after three transfusions on July 2nd, July 17th and July 19th. She bled from every cut surface and died that night.

Hysterectomy was done in 3 cases of bilateral cancer of the ovaries, and once each in a ruptured cornual pregnancy, in a placenta accreta, in a badly infected uterus without morbidity, and in a moribund term uterus ruptured hours before admission to hospital.

We had 21 cases of complete uterine prolapse, with no mortality. We seldom do hysterectomy for genital prolapse, for which we have been very partial to the so-called Manchester or Fothergill operation, and have for years wondered why it is not more popular with American gynæcologists. Recently, it has been more widely used. We reserve hysterectomy for the cases of complete uterine prolapse where the Mayo type vaginal hysterectomy with vaginal plastic is indicated. One case is worthy of comment.

Mrs. O.V., aged 48, reported that she had had one full term pregnancy 18 years before and an abdominal cyst that was aspirated three months later. She had a complete uterine and vaginal prolapse with a cystic tumour, the size of a seven months' pregnancy, in the abdomen. Cystoscopic examination showed a huge right hydronephrosis. A vaginal hysterectomy with vaginal plastic and a right nephrectomy were done under gas anæsthesia, at one sitting, without complication.

The complete record of all our cases is as follows. There were 321 total hysterectomies including 36 vaginal hysterectomies with a mortality of 2. There were 287 supra-vaginal hysterectomies with a mortality of 7. There were 129 myomectomies without mortality. Total 741 cases with a mortality of 9.

THE SIZE OF THE HEART AFTER CORONARY THROMBOSIS*

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THE part played by high blood pressure in causing enlargement of the heart in any group of patients over forty years of age is considerable. It is, indeed, the general practice of pathologists to ascribe to hypertension all cases of cardiac enlargement in which is found "a left ventricular hypertrophy of high degree that is not associated with valvular disease, adherent pericardium, syphilitic aortitis, chronic glomerulonephritis or hyperthyroidism".[4] The clinical diagnosis of these conditions, except adherent pericardium, which so seldom alone causes enlargement,[30] is not usually difficult, and the pathological dictum is largely applicable during life, particularly when the height of the blood pressure is actually known.

Readings at or above an arbitrary hypertensive level of 160 mm. of mercury systolic and/or 100 mm. diastolic before or after the attack were actually recorded in more than 70 per cent of a series of cases of coronary thrombosis recently analyzed by the author,[28] and from which were drawn the group on which the present paper is based.

Coronary thrombosis, apart from that due to syphilitic aortitis, is from the point of view of pathology merely an episode in coronary arteriosclerosis (atheroma). When we consider, therefore, the incidence of cardiac enlargement in coronary thrombosis it is necessary to inquire whether or not coronary sclerosis can of itself, in the absence of hypertension, bring about an increase in the size of the heart.

Lewis and Drury[19] during their investigation into the problem of arteriovenous aneurysm concluded that deficient blood supply came under suspicion as a cause of the cardiac enlargement associated with constrictive lesions of the coronaries. Smith[34] found that ligation of the coronaries in dogs resulted in ventricular hypertrophy, and later Smith, Miller and Graber[35] were satisfied from their experiments on blood pressure variations in the coronary system that decreased coronary flow was a factor in the development of hypertrophy. On the other hand Sutton and Davis[38] failed to find increase in the size of the heart in dogs after coronary ligation followed by exercise, and came to the conclusion that "myocardial infarction *per se* does not cause hypertrophy". There is experimental[13] and clinical[1, 32] evidence to show that anæmia can lead to cardiac enlargement; and enlargement is the rule in those cases where venous blood is supplied to the myocardium by an anomalous coronary artery arising from the coronary sinus.[10, 6] Bouchut and Froment[3] discuss at length lack of oxygen in tissues as a cause of hypertrophy.

The various hypotheses regarding the mechanism of hypertrophy scarcely appertain to this clinical study. It is perhaps pertinent, however, to recall the conception generally favoured by physiologists today, namely, that dilatation and stretching of the muscle fibres always precedes hypertrophy.[16] Extra work done by the heart *within the limit of its reserve* does not cause hypertrophy, this being true whether the heart muscle is healthy or diseased; it is only when the myocardium labours constantly *at the limit of its reserve* that the dilatation necessary to produce hypertrophy occurs.[7] This dilatation may be either physiological or pathological,[25] depending on whether a healthy muscle is involved in mechanical difficulties or whether there is decreased muscle tone through disease. Wiggers[39] has pointed out that small grades of dilatation (such as would precede hypertrophy) can only be studied in the living heart, and that inability to recognize them *post mortem* is not a reason for denying their existence.

As there is evidence that in some conditions an insufficient blood supply to the myocardium over a reasonably long period of time may result in hypertrophy, there seems no reason for denying the possibility that it may also be

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induced as a result of lessened circulation in the coronary vessels due to severe sclerosis. It is necessary to emphasize, however, that hypertrophy after coronary thrombosis is dependent for its development on the state of the remaining *living* musculature and the demands made upon it. The fibrosis resulting from infarction can be a factor only in so far as it reduces the muscle mass to such an extent that the extra work thrown on the remaining myocardium lessens its reserve appreciably. McCallum[21] states that the ventricular myocardium may be almost completely sclerosed by repeated injections of alcohol before it begins to fail.

A survey of the literature suggests that cardiac hypertrophy as a result of coronary disease does occur, but that it is not common. Investigation from the pathological side is complicated by a frequent inability to gauge correctly the effect of other factors known to cause hypertrophy, particularly hypertension. Delafield and Prudden[11] merely state that hypertrophy may follow or be associated with chronic interstitial myocarditis (fibrous hyperplasia). Smith and Bartels[36] described two cases, with necropsy, of gross hypertrophy associated with myocardial infarction and normal blood pressure in young persons aged 35 and 36 years. They later[3] reported 37 more cases in which they considered that all the conditions known to cause cardiac hypertrophy had been satisfactorily excluded. Prior blood pressure readings over a period of years were available in a few, but they admitted the possibility of prior hypertension in others with one reading only. In 113 fatal cases of coronary sclerosis with necropsy Nathanson[26] could find no relation between the degree of fibrosis and the weight of the heart. Miller and Weiss[24] reported 19 necropsies showing gross areas of myocardial fibrosis (cases of recent infarction were excluded) all of which had unhypertrophied hearts. Bell and Clawson[4] have contended that, physiologically speaking, it is unsound to argue that coronary disease produces hypertrophy, and that it may actually prevent further enlargement in an already hypertrophied heart by bringing about a decrease in the blood supply to the myocardium. The findings of Nemet and Gross[27] in 100 necropsies in arteriosclerotic heart disease made it difficult for them to believe that the myocardial fibrosis of coronary disease is an adequate cause for the development of hypertrophy, although they admitted its possibility from a physiological point of view.

From the clinical side Parkinson and Bedford[31] have stated that "coronary disease alone can cause some hypertrophy of the heart, especially of the left ventricle, but unless high blood pressure or valvular disease is present in addition cardiac enlargement is not a feature". They added that when the heart is really large hypertension preceding the attack should be suspected. Willius[40] reported a case in which enlargement of the heart was known to have developed at some time during the seven years following an attack in the absence of hypertension, and stated that "cardiac hypertrophy frequently follows cardiac infarction, which may become marked if multiple infarctions have occurred". Negative evidence is presented by Horine and Weiss,[15] who observed that in 20 cases of coronary thrombosis followed radiologically for periods ranging from five months to almost ten years after the attack the heart was of normal size at the first and at all subsequent examinations.

The incidence of cardiac enlargement in coronary thrombosis.—Literature on the incidence of cardiac enlargement following coronary thrombosis is based chiefly on the weight of the heart at necropsy. Difficulty in defining the upper limit of normal, and the consequent assumption by pathologists of somewhat different criteria for its determination have rendered strict correlation of all published data impossible. Barnes and Ball[2] and Lisa and Ring[20] found 86 per cent and 77 per cent of hearts in their respective series to weigh more than 400 grams; and Levine[18] found hypertrophy in 89 per cent, when weights of 350 grams and 400 grams were taken to be the upper limits of normal for females and males, respectively. While their criteria are not stated, almost constant enlargement was discovered at necropsy by Evans, Ambler and Dodson,[12] and an incidence of more than 80 per cent by Meakins and Eakin.[23]

Arbitrary standards cannot be set up for the clinical determination of enlargement, and there will always be a certain number of cases in which "enlargement doubtful" must describe the result of radiological examination. If

400 grams be accepted as the upper limit of normal heart weight it follows that in order to be clinically certain of enlargement weights considerably in excess of this must obtain. Of hearts in coronary thrombosis weighing more than 500 grams Barnes and Ball found only 51 per cent, and Lisa and Ring 57 per cent. It might be predicted that figures showing the incidence as determined by x-ray would be somewhat larger than this, always providing that hypertrophy rather than dilatation is responsible for the enlargement. It will be shown that in the series now to be examined dilatation played a part considered to be almost negligible, and that the incidence of enlargement closely approximated to the expected figure.

THE AUTHOR'S SERIES

The following clinical observations on the incidence and cause of cardiac enlargement are based on an analysis of 200 cases which had survived the attack by at least three months. The series was entirely drawn from, and comprised almost 95 per cent of, a group analyzed by the author with reference to blood pressure findings[28] and prognosis[29] after coronary thrombosis. The only selection made was such as to ensure that in every case the size of the heart had been judged radiologically, and all reference to determination of cardiac size in the present series implies the use of this method only. It invariably consisted of fluoroscopy in the antero-posterior and right and left oblique positions, frequently supplemented by orthodiagram and/or teleroentgenogram. It has been the practice in the Cardiac Department of the London Hospital to base the diagnosis of enlargement on the observer's judgment after inspection of the heart in the three standard positions; it is felt that this is the best method for routine clinical use, and that by it an accurate estimate of the relative sizes of the various chambers can be made.

Of the 200 patients, 144 (72 per cent) had more than one x-ray examination, and 40 (20 per cent) were observed at intervals during more than five years. The average period of time elapsing between the attack and the last record of size was almost three years. Cardiac enlargement was found in 128 (64 per cent). For purposes of this communication 32 cases in which the report was "enlargement doubtful" were not included in this percentage, but were classified as normal.

CAUSES OF ENLARGEMENT

Incidental enlargement.—Three cases of valvular disease and one in which hyperthyroidism had recently been diagnosed were placed in an *"incidental enlargement"* group. These conditions were for purposes of classification considered to be the sole cause of the enlargement, though two of those with valvular lesions had high blood pressure.

Hypertension.—High blood pressure was considered to be the sole outstanding cause of enlargement in 106 cases (82.8 per cent). Hypertension had actually been recorded by the sphygmomanometer in 100 of these, either before or after the attack; it was presumed to have been present in the other 6, two of whom had the enlargement prior to the attack without its progressing subsequently, while 4 gave borderline readings on several occasions.

Arteriosclerosis.—So far as is known none of the cases placed in the arteriosclerotic group was hypertensive.

Bundle-branch lesions.—It has frequently been observed by Parkinson and his co-workers that bundle-branch lesions may be associated with cardiac enlargement in the absence of hypertension. In these cases it is probably correct to regard the onset of the bundle-branch lesion as an indication of coincident aggravation of the coronary disease, and to assume that the resulting myocardial ischæmia is the important factor in causing enlargement. It is possible that alteration in the mechanism of the heart beat owing to the disorder of conduction may also play a part, but direct evidence on this point is lacking.

Only 3 cases of the present series were placed in this sub-group, the bundle-branch lesion having been observed in two of them to arise some months following the attack, and more or less coincidentally with the increase in size of the heart. Bundle-branch block was found in 5 other cases in association with hypertension, as well as in 1 case of cardiac aneurysm; in all of them except one the heart was enlarged.

Cardiac aneurysm.—Enlargement due to this condition may be classified as resulting from the underlying sclerosis, and may also be cited as

directly caused by the thrombosis *per se*. Figures are lacking to show the frequency with which cardiac aneurysm has been recognized clinically, and it is safe to assume that without radiological aid the diagnosis cannot be made with any degree of certainty. Its incidence at necropsy has been found by Parkinson and Bedford,[31] Levine,[18] and Lisa and Ring,[20] to be about 6 per cent; Benson and Hunter[5] placed it rather higher (9.5 per cent).

In the present series aneurysm was diagnosed radiologically in 4 cases, confirmed at necropsy in one. It is interesting to note in them all the absence of hypertension following the attack; unfortunately in none was the prior height of the blood pressure known.

Chronic myocardial ischæmia.—By process of elimination it was concluded that in 4 cases coronary arteriosclerosis, by bringing about a decrease in the blood supply to the myocardium, was alone the cause of enlargement. That hypertension played no part is proved by knowledge that in each case the blood pressure was low both before and after the attack, and that the heart was normal in size at some time during the few months following it. The latter observation probably rules out in addition the possibility that the enlargement discovered later was due to cardiac aneurysm. It is well recognized that the radiological detection of this latter condition is difficult, but it should not be so where the aneurysm is of sufficient size to be the sole cause of an appreciable enlargement.

Cause of enlargement uncertain.—

Seven cases remained in which the factor or factors causing the increase in size could not with reasonable certainty be determined. The enlargement was discovered at the first radiological examination, which took place in all except one patient within 6 months of the attack. Four, followed further, failed to show progressive enlargement and prior hypertension must here be suspected. One showed increase in size following a second attack of coronary thrombosis 18 months later. No other case was found in which the subsequent attack might have been considered a factor in causing enlargement.

TABLE I

THE SINGLE OR PREDOMINANT CAUSE OF ENLARGEMENT OF THE HEART IN 128 OF A SERIES OF 200 CASES OF CORONARY THROMBOSIS STUDIED RADIOLOGICALLY

Cause of enlargement	*No. of cases*	*Percentage of cases in whole series (200 cases)*	*Percentage of cases of enlargement (128 cases)*
Incidental enlargement ..	4	2	3.1
Hypertension	106	53	82.8
Coronary arteriosclerosis.	11	5.5	8.6
Bundle-branch lesions .	(3)	(1.5)	(2.4)
Cardiac aneurysm	(4)	(2)	(3.1)
Chronic myocardial ischæmia	(4)	(2)	(3.1)
Undetermined	7	3.5	5.5
Totals	128	64	100

DEVELOPMENT AND PROGRESSION OF ENLARGEMENT UNDER OBSERVATION

In 20 cases the enlargement was found to arise or progress during the period of observation. In this group 8 are known to have had normal-sized hearts when they were first examined radiologically; 4 of them were non-hypertensives in whom the enlargement was considered due to coronary sclerosis itself (chronic myocardial ischæmia). Two others are known to have had slight hypertension but no enlargement 5 months and 9 months, respectively, prior to the attack; they both continued with mild hypertension subsequent to it, but it is possible that chronic myocardial ischæmia as well played a part in the development of enlargement. Of the 2 remaining one showed no enlargement 3 weeks after the attack; 18 months later he had a second attack, in which pericardial friction was detected; after a further six months his blood pressure was 215/115, and he had nocturnal dyspnœa and enlargement of the left ventricle. The other patient had, three years after the attack, some intraventricular block with doubtful enlargement; four years later he developed partial heart block and the heart was then found to be moderately enlarged. During the next two years there was no further increase in size, although the systolic blood pressure remained above or in the neighbourhood of 200 until his death three years later in a Stokes-Adams attack.

Twelve patients had enlarged hearts when they were first seen. Three were non-hypertensives already referred to, one having acquired increase in the degree of enlargement subsequent to a second attack of coronary thrombosis, and the other two following the observed onset of bundle-branch block. Six were hypertensives with consistently high

blood pressure, and in 2 of them the increase in enlargement was noted following the onset of bundle-branch lesions. In the remaining 3 the increase in enlargement was associated with congestive heart failure.

Congestive failure associated with increase in the degree of enlargement.—

Parkinson, in his Lumleian lectures,[30] has pointed out that changes in the heart shadow during congestive failure are chiefly of shape and position rather than of size, but that distension of the superior vena cava and right auricle may occur. Failure was observed in 24 of the series. In 16 of these radiological observations were made during the stage of œdema, as well as before its onset or after it had cleared up; in only 3 was any definite change in size observed. Here enlargement, which was already present, increased during failure, the cardiac rhythm remaining normal; in 2 of them œdema persisted to a fatal termination, and no further observations on the size of the heart were made; in the third there was recession to approximately the "pre-failure" size with the clearing up of the congestion.

Temporary enlargement of the heart shadow appearing early after the attack.—

Dilatation of the heart, appreciable on x-ray examination, has been described by Jegorow[17] and Frank[14] as occurring in a few cases during the course of the two or three weeks immediately after the attack, followed by an early return to normal size. Wolff and White[41] also refer to dilatation that "rapidly occurs and can be watched as it definitely develops". Roesler[33] mentions the occasional occurrence of acute dilatation in coronary thrombosis.

Wollheim[42] was apparently the first to recognize during life a pericardial effusion complicating cardiac infarction, but unfortunately he fails to state on what grounds the diagnosis was made. Master and Jaffe[22] have reported two cases based on radiological evidence, and believe that its occurrence is not rare. It has been found at necropsy in association with recent infarction by Levine,[18] Smith and Willius,[37] and Camp and White.[9]

In none of the present series was either of these two uncommon conditions observed. Eleven patients were x-rayed within a week of the attack, a further 16 within a month, and in all of these the size of the cardiac shadow was considered to be normal. It is admitted, however, that the number is small, and the fact that the series contains only cases that survived the attack by three months must be borne in mind.

Incidence of normal-sized hearts following coronary thrombosis.—

That the heart may remain normal in size for long periods following an attack of coronary thrombosis has been shown by radiological methods as well as by necropsy findings.

In the present series 72 patients (36 per cent), including 32 reported as having doubtful enlargement, were considered to have normal-sized hearts. In them the average period elapsing between the attack and the last x-ray was more than three years; in 18 it was more than five years. It is worthy of note that in seven of the above group the last x-ray observation was subsequent to one or more further attacks of coronary thrombosis.

SUMMARY

1. Enlargement of the heart was found by radiological methods in 64 per cent of a series of 200 patients who had survived an attack of coronary thrombosis. Doubtful enlargement, 16 per cent of the series, was for the purpose of this paper added to the normal group, but the high incidence of enlargement in published necropsies favours the view that these doubtful hearts are in reality enlarged.

2. The factors causing enlargement after coronary thrombosis are discussed. By far the most important proved to be hypertension, which was held to be the single or predominant cause in more than 80 per cent of all cases with enlargement.

3. Disease of the coronary arteries, either the actual thrombosis with its resulting infarction or the underlying arteriosclerosis, led to increase in the size of the heart in a total of 11 cases (8.6 per cent). Of these, 4 (3.1 per cent) had cardiac aneurysm, 3 (2.4 per cent) had a bundle-branch lesion, and the remaining 4 (3.1 per cent) had enlargement, apparently due to chronic myocardial ischæmia alone.

4. No example of so-called acute dilatation of the heart was discovered among 27 patients examined radiologically within a month of the attack. Congestive failure, which was not seen in hearts of normal size, seldom led to an

appreciable increase in the degree of enlargement.

5. About a third of the patients (36 per cent) failed to show or to develop enlargement, though watched over periods averaging more than three years following the first attack of coronary thrombosis; and this number included several with recurrent attacks.

I am deeply indebted to Dr. John Parkinson, Physician to the Cardiac Department of the London Hospital, for placing his private case-notes and x-ray records at my disposal, and for his advice in the preparation of this paper.

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Reprints with the complete list of references can be obtained from the author.

EXPERIMENTAL ARSPHENAMINE DERMATITIS: THE INFLUENCE OF VITAMIN C IN THE PRODUCTION OF ARSPHENAMINE SENSITIVENESS*

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THERE are but few statements in the literature regarding the influence of vitamin C in the production of arsphenamine dermatitis or its effect on the course of existent dermatitis. Chapman and Morrell,[1] in an initial group of experiments, stated that guinea pigs on a diet high in vitamin C developed marked sensitization reactions to neoarsphenamine, while pigs on a low vitamin C diet developed a less marked sensitivity. Sulzberger and Oser,[2] however, reported findings which suggested that a high vitamin C diet inhibited the development of sensitivity. The pigs showing the most marked sensitization reactions were often those with the most florid scurvy. In a later communication Chapman[3] stated that further experiments had indicated that there is a certain level of vitamin C concentration above which no reactions occur. He also believed that the concentration of vitamin C has nothing to do with the development of sensitivity, but affects only the reaction following the provocative dose of neoarsphenamine (the second intradermal injection, given 28 days after the initial injection). He interpreted this result to mean that although sensitization had developed its existence could not be proved by a second provocative intradermal injection. Dainow[4] has recently reported that vitamin C given intravenously definitely shortened the course of three cases of post-arsphenamine dermatitis.

In former experiments the author pointed out that arsphenamine sensitiveness, as it occurs in guinea pigs, is only roughly comparable with that seen in man. In the guinea pig an apparent locally irritating effect of a second intradermal injection, given 28 days after a first injection, is necessary for the completion of the sensitization cycle. In neither guinea pigs nor human beings has conclusive proof been put forth which demonstrates that arsphenamine idiosyncrasy is a true allergic phenomenon. In a later paper further studies will be reported in regard to the allergic status of the dermatitis; in this communication supposedly irritant effects on the individual cell will be studied. These effects consist wholly in artificially changing the vitamin C content of the diet in different groups of guinea pigs.

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MATERIALS AND METHODS OF THE PRESENT STUDY

Young adult guinea pigs weighing about 300 g. were selected. The scorbutogenic diet recommended by Demole[5] was used. Cod liver oil was fed by mouth to each pig in a dosage of 0.2 g. once weekly. The abdomens of the pigs were denuded of hair previous to all intradermal testing with a paste consisting of 50 per cent barium sulphide in starch. All dosages of neoarsphenamine were uniform, the 0.1 c.c. dose being prepared so as to contain 0.00015 g. of neoarsphenamine. This neoarsphenamine was of a batch previously shown to have a high sensitization index in a group of ten normal guinea pigs. In normal pigs when sensitivity to neoarsphenamine has developed the first injection results in a small hard papule, appearing in from six to sixteen days, and the second injection in an erythematous flare, usually occurring within twenty-four hours. The two injections were given 28 days apart, to allow for the development of sensitivity.

Experiment 1. — Thirty normal guinea pigs were placed on the scorbutogenic diet, without supplementary vitamin C, for one week. The reason for this preliminary interval was to bring the vitamin C concentration in each pig to a low level, for Youmans and his co-workers[6] have shown that vitamin C is stored in the body and a low intake does not result in an actual deficiency until this reserve is depleted. At the end of the seven-day period varying amounts of vitamin C (cevitamic acid), diluted in a suitable amount of water, were given by pipette to each pig. Five pigs were given 0.025 mg.; five 0.05 mg.; ten 0.1 mg. and ten 0.2 mg. These dosages were continued daily throughout the 28-day test period. On the first day neoarsphenamine was injected intradermally in the abdomens of all pigs in the standard dosage. At the end of the 28-day interval neoarsphenamine was reinjected on the opposite side of the abdomens.

Results.—Of the first group of 5 pigs, 4 reacted to the first injection. Small papules appeared on the eighth day after the injection in one pig, on the ninth day in two pigs, and on the eleventh in one. In each instance the papules were small, reached a maximum of 5 to 7 mm. in diameter in about ten days and then slowly receded. There were no œdematous flares. Unfortunately, none of this group of pigs lived to be retested. One died on the 9th day, and might have developed a reaction at a later date if it had lived. A second pig died on the 18th day. Two died on the 20th day and one on the 27th. Autopsies revealed marked malnutrition, avitaminosis, and a complicating pneumonia.

Of the second group of pigs only two had reactions to the first injection. A small papule developed on the 10th day in one pig and persisted until the death of the pig four days later. In the second pig a small papule developed on the 9th day, reached a maximum on the 15th day, and then slowly subsided. None of this group of pigs lived to be retested. In addition to the death mentioned above two pigs died on the 15th day, one on the 20th, and one on the 28th. All of these pigs were greatly overweight, showed gross signs of scurvy, and had a complicating pneumonia.

TABLE I.

NUMBER OF PIGS REACTING

Group 3:— (In Days after first Injection) Second Injection ∨

Reactions	Days 8	9	10	11	13	14	15	17	18	20	21	22	24	27	28	29	31
±	2	1	1	1						1	1	2	1	4			
+	1	2	2	3	5	5	5	3	2	2	2	3	3				
++		1	1	1				2	3	3	2	3	3		1		
+++					1	1	1								1	(4+)	1

Group 4:—

Reactions																	
±			3	2	1	1	1	1	1	2	2						1
+		1	2	1	1	1	1	2	2	1	2	3	3	5	1		
++				2	2	2	2	2	3	4	4	4	4	2	1	3	5
+++					1	1	1	1							4	3	1

Reactions are tabulated above as ±, questionable erythema; +, 6 mm. in diameter (later becoming papular); ++, 12 mm. in diameter erythema > papule; +++, quarter-sized erythema, often with small central œdema; ++++, silver dollar-sized (or larger) erythema with large area of central œdema.

There were 10 pigs in the third group, 6 of which developed reactions. As will be seen in Table I, the maximum intensity of the reactions was not reached until the 20th day after the initial injection. Only 3 of these pigs lived to be retested, one dying on the 13th day, one on the 16th, two on the 22nd, one on the 23rd, one on the 24th and one on the 28th. The results in the three retested pigs were interesting. One pig developed a small papule on the 7th day after the first injection which reached a diameter of 14 mm. on the 14th day, at which time it was definitely œdematous. It then gradually subsided, but a small papule was still present on the 28th day. This pig *did not react* to the second injection. The second pig,

which had shown a two-plus reaction to the first injection, developed a large erythematous reaction (13 by 25 mm.) ten hours after the second injection. Two days later the reaction became hæmorrhagic, then gradually subsided. The last reacting pig, which had shown a two-plus reaction to the initial injection, developed a large œdematous lesion (15 mm. in diameter) eight hours after the second injection. In addition there was a small area of erythema at the site of the first injection.

Nine of the fourth group of 10 pigs lived to receive the second intradermal injection, and were of value from the standpoint of comparative results. As will be seen in Table I, seven of these pigs developed reactions to the first intradermal injection. The maximum intensity was not reached until the 22nd day. A minute papule was seen on one pig from the 20th to the 22nd day, but there was no reaction to the second injection and the papule was not considered significant. A small papule appeared in one other pig on the 20th day and this pig reacted strongly to the second injection. The 7 initially reacting pigs all had local lesions following the second injection. The maximum intensity of the lesions was not reached until 48 hours after the injection. One pig did not react until after 72 hours. In one instance a reaction occurred similar to that seen in one of the pigs in group three. This was a slight œdema in, and a zone of erythema around, the still present reaction to the first injection.

Comment.—It is impossible to compare the first three groups with the fourth group because of the failure of the pigs to live until the second injection could be given. Judging by the sensitization flares following the first injection, however, it seemed reasonable to assume that sensitization developed about as easily in the group receiving 0.2 mg. of cevitamic acid as in those receiving 0.05 mg. or less. Six of the 10 pigs receiving 0.05 mg. or less developed reactions, while of the group receiving 0.2 mg., 7 reacted. In the intermediate group, receiving 0.1 mg., 6 out of 10 pigs reacted. The size of the initial injection did not vary greatly throughout the series. The reaction was large and œdematous in 3 pigs. Two of these pigs were in the group receiving 0.2 mg., while the other pig had received 0.1 mg. daily. Emphasis should be placed on the indolence of the lesions, which was not seen in previous experiments. Many of the initial lesions persisted until the second injection was given, and in two instances the old lesions were exacerbated by the second injection. The reactions to the second injection were somewhat larger than usually seen in the normal animal, and they persisted for three days or more in five instances. One curious feature was the failure of one pig, which had previously shown a large œdematous reaction to the initial injection, to react to the second injection. This pig must have been sensitive, yet it did not react. Sulzberger has called this phenomenon "cachectic energy".

Conclusions.—(1) Variations in the vitamin C content of the diet, ranging from 0.025 mg. to 0.2 mg. daily, had little if any influence upon the development of cutaneous arsphenamine sensitiveness in guinea pigs. (2) A low vitamin C diet resulted in more indolent and more severe reactions than were previously encountered in a series of normal guinea pigs.

Experiment 2.—An experiment was performed to determine the effect, if any, of large doses of vitamin C in inhibiting the reaction to the second intradermal injection of neoarsphenamine. Twelve young adult female guinea pigs, each weighing about 300 g. were placed on the vitamin C deficient diet. Neoarsphenamine in the usual dosage was given to each pig at the start of the experiment. One week later each pig was given by pipette 0.2 mg. of cevitamic acid daily. This dosage was continued for two weeks; it was then increased to 20 mg. daily for the one week preceding the second injection of neoarsphenamine.

TABLE II.

NUMBER OF PIGS REACTING

(In Days after first Injection)								Second Injection	
	Days							V	
Reactions	*9*	*11*	*12*	*14*	*18*	*22*	*26*	*28*	*31*
±		1	1	1					
+		4					8	2	
++	1	2	8	7	4	7		2	8
+++				1	5	3		4	1

Results.—The reactions, as can be seen in Table II, were somewhat delayed in their appearance, but were much larger than the usual reaction to the initial injection of neoarsphenamine. One of the lesions was œdematous, while in other pigs nodules rather than papules were present. Of note was the fact that one of the pigs not reacting to the first dose developed a pea-sized papule 72 hours after the second

injection. Another pig which had shown a two-plus reaction to the first dose did not react to the second. Two pigs were negative throughout.

Conclusions.—(1) A low vitamin C diet increased the intensity of the initial reactions and retarded their involution. (2) There seemed to be very little inhibiting effect of large doses of vitamin C for one week on the reactions to the second injection, except for some delay in the appearance of the lesions. After sensitivity had developed large doses of vitamin C did not prevent the appearance of the subsequent reaction.

Experiment 3.—Nine guinea pigs were chosen for this experiment. All had shown strong intradermal reactions to neoarsphenamine in the spring of 1935 and negative reactions in the following summer. The standard scorbutogenic diet was given for the first two weeks of the experiment; during the last two weeks 0.05 mg. of cevitamic acid was given daily to each pig. Neoarsphenamine in the usual dosage was given at the start of the experiment.

Results.—Four of these 9 pigs developed immediate reactions to the initial injection, 2 being two-plus and 2 three-plus reactions. Because of the severe degree of scurvy induced only two pigs lived to be reinjected after the 28-day interval. One of these had shown a two-plus and the other a three-plus reaction to the initial injection. Both pigs developed four-plus reactions to the second injection.

Conclusion.—Four out of 9 pigs, all previously sensitized, but all non-reactive in the summer of 1935, showed the immediate type of reaction characteristic of existing sensitivity. Some unknown factor, possibly the avitaminosis, had intervened to reactivate the previous sensitivity.

Experiment 4.—A group of 14 pigs in which cutaneous sensitization to neoarsphenamine could not be produced in the summer of 1935 were placed on the standard scorbutogenic diet. Neoarsphenamine in the usual dosage was given at the start of the experiment. Cevitamic acid, 0.05 mg. daily, was given to each pig after a two weeks' period. All of the pigs in this group died before retesting could be done, but the reactions to the initial injection were of great interest and deserve special comment.

Results.—Three of this group of 14 pigs developed three-plus reactions within 24 hours after the injection. In a fourth pig a two-plus reaction appeared after 5 days and became four-plus after 2 more days. Four other pigs developed small papules on the seventh day after the injection.

Conclusions.—(1) A previously silent arsphenamine deposit was apparently reactivated, possibly by the low vitamin C diet, causing immediate sensitization reactions in 3 pigs. (2) Delayed reactions characteristic of developing sensitivity appeared in 4 other pigs, indicating an increased ease with which cutaneous reactivity to neoarsphenamine was developed.

Experiment 5.—Twenty-two young adult, normal, male pigs were placed on the scorbutogenic diet. Huge doses of cevitamic acid, 50 mg. per pig per day, were given throughout the 28-day period. Neoarsphenamine in the usual dosage was injected at the onset of the experiment and again after a 28-day interval. There were no reactions to either the initial or to the second test dose of neoarsphenamine. The same diet was then continued, but all cevitamic acid was withheld for a four weeks' period. Neoarsphenamine was then injected intradermally two weeks after the vitamin C was discontinued.

Results.—Seven of these 22 pigs developed immediate reactions to the injection, suggesting that a shift from a high to a low vitamin C diet had reactivated a previously silent arsphenamine deposit, resulting in reactions indicating an existing sensitivity to neoarsphenamine.

Conclusion.—A high vitamin C diet does not, apparently, prevent the development of cutaneous sensitization to neoarsphenamine but merely inhibits the cutaneous reactions. Evidence of previous sensitization can be obtained by sharply decreasing the vitamin C content of the diet and then retesting with neoarsphenamine.

SUMMARY AND CONCLUSIONS

1. Variations in the vitamin C content of the diet, ranging from 0.025 to 0.2 mg. of cevitamic acid per day, had little if any influence on the development of cutaneous arsphenamine hypersensitiveness in guinea pigs. At the lower levels of vitamin C diets death from scurvy was common.

2. A low vitamin C diet resulted in more indolent and more severe reactions than was previously encountered in a series of normal guinea pigs.

3. A low vitamin C diet increased the intensity of the reactions to the initial dose of neoarsphenamine and retarded their involution. Large doses of vitamin C, given for one week before the second test injection, had little inhibiting effect (except for some delay in the appearance) in the intensity or frequency of the reaction to the second intradermal injection.

4. A low vitamin C diet given to a group of formerly sensitive pigs, which, however, were unsensitizable during a subsequent summer period, resulted in immediate flare reactions to intradermally injected neoarsphenamine in 4 of a group of 9 pigs.

5. In another group of 14 pigs, in which cutaneous sensitization to neoarsphenamine could not be produced in the summer of 1935, a vitamin C-deficient diet for a two-week period resulted in reactions of existing sensitization in 3 pigs and of developing sensitization in 4 other pigs.

6. The *modus operandi* of the vitamin C-deficient diet is unknown; it apparently reactivated a silent arsphenamine deposit in one group of previously unsensitizable pigs and rendered the cutaneous cells susceptible to the development of sensitivity in another group.

7. Twenty-two pigs which had been given huge doses of cevitamic acid could not be sensitized to neoarsphenamine. The nature of this inhibiting action of vitamin C could not be determined. Either the development of sensitization was prevented or the cells were both hypersensitive and immune.

8. Seven of these 22 pigs, all of which had been placed on a vitamin C-deficient diet for two weeks, then developed immediate sensitization reactions to subsequently injected neoarsphenamine.

9. On the basis of the above experiments a high vitamin C diet should be of value to all patients receiving an arsphenamine, and particularly to those who had inadvertently received a paravenous injection.

10. Intensive therapy with cevitamic acid may be of value in patients with post-arsphenamine dermatitis, and to such patients at a later date if further therapy with an arsenical is to be attempted.

NOTE.—The author is greatly indebted to the Abbott Laboratories, Limited, of Montreal, for their kindness in supplying the cevitamic acid. Without their generous cooperation this work would not have been possible.

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ŒSTROGENIC HORMONES IN THE TREATMENT OF VULVO-VAGINITIS IN CHILDREN*

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VULVO-VAGINITIS of childhood has been recognized as a clinical entity for many years, and its etiology and treatment are matters of considerable controversy.

As early as 1877 Suchard recognized the epidemic nature of vulvo-vaginitis and reported a series of cases of gonococcal origin. Later, the literature contained enthusiastic reports on the remarkable frequency with which the gonococcus was considered as an etiological factor. Fraser stated that in his experience the incidence of such infections was so high that all cases of vulvo-vaginitis should be looked upon as gonococcal in origin until proved otherwise. Stein[8] and Williams,[9] claim that from 50 to 60 per cent of all infections of the lower genital tract in female children are due to the gonococcus. Ruys[7] places this figure at 20 per cent, and believes true gonococcal infections to be rare. It is agreed by many authorities that the types which are acute in character and epidemic in distribution are unquestionably gonococcal. The disease however, has a notorious chronicity, and it becomes increasingly difficult to demonstrate the organism at this stage, so that the casual clinician is often misled in his diagnosis. A more detailed discussion of the bacteriological aspect is quite beyond the scope of this presentation.

During the past few years our ideas regarding the treatment of vulvo-vaginitis in children have changed materially. This is ascribed to a more careful study of the pathological lesions produced. Very early in the course of an acute

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gonorrhœal infection the elusive gonococcus penetrates the delicate lining membrane of the vagina and a subepithelial or submucosal infection is established. In such a location the organism is protected from local antiseptics. Robert Lewis,[4] working on the basis of Edgar Allen's observations of the effects of the œstrogenic hormones on the genital tract of immature monkeys, first suggested the treatment of gonorrhœal vaginitis by hormones. A new field of therapeutics was thereby opened up, namely, the use of hormones to produce an alteration in the tissues in such a way that infection may be eradicated. Lewis demonstrated clinically the rationale of hormonal therapy, and published a series of cases of vulvo-vaginitis which had been treated by this method. Since then the literature contains several contributions on the efficacy of this treatment, and among these are the papers of Miller,[5] Huberman and Israeloff,[3] Hartman.[2]

The apparent simplicity of this treatment and the favourable reports offered by many of the authors might lead one to the hasty conclusion that an infallible cure is easily obtained. We must maintain a conservative attitude in this respect and from a physiological aspect assure ourselves that there is no deleterious effect on the reproductive and endocrine glands. There has probably not been sufficient time or means at our disposal to accurately determine this fact. Allen noted that associated with metaplasia of the vaginal mucosa there was a development of the cervical glands and invasion of their lumina by polymorphonuclear leucocytes. He also stated that the endometrium was built up to a mid-interval stage. Novak,[6] in a recent publication, states, "The physiological effect of œstrogenic substances is to produce growth and hyperæmia, but not secretory activity." Hartman is of the opinion, based upon experimental work with animals, that the dosage of this hormone sufficient to produce vaginal reaction is far less than that which would lead to injurious changes or uterine bleeding. Despite the fact that hypertrophy and increased vascularity of the labia have been noted, I am not aware that any cases of true uterine bleeding have been reported.

Hartman and Novak are agreed that the ovary does participate in the reaction, and state that there may be an inhibition of growth in the follicle, but such effects are only temporary and no damage to the ovary results. Miller, in one instance, removed a section of the ovary from a nine-year old child after the injection of 1,000 Doisy rat units of theelin, and, on histo-pathological examination, found the tissues had not been affected in any way. The possibility of producing uterine or tubal extensions of the infection as a result of pelvic hyperæmia is more apparent than real, although only continued observation of these cases can determine this point. The rapidity and permanency of cure varied considerably with each individual investigator, partially because of different preparations being used and partially because of different modes of administration being adopted. It is, further, agreed that the acute cases of gonococcal infection with profuse discharge, and which develop early complications, do not respond so readily as do the mild types.

Lewis originally reported a series of 8 cases, with resultant cure in every instance, averaging 21 days. Huberman and Israeloff reported 6 cases, with cures in six to nine weeks, using "amniotin". James Miller[5] presented probably the largest series of cases and reviewed a two years' experience, using the same œstrogenic substance as was used in the series to be reported. He observed 42 cases for a period of 6 months, and 23 for a year after treatment. I find that, calculating his results on a percentage basis of these two series, good results vary from 45 to 68 per cent (averaging 56); temporarily controlled, from 21 to 38 per cent (averaging 29); unsatisfactory, or failures, 10 to 15 per cent.

The present series of 25 cases were observed and treated in the Outdoor Department of the Children's Memorial Hospital in Montreal. This work was undertaken in September, 1934, and the entire group was last checked up in June, 1936. The series was divided into two groups for the purpose of classification and comparison. Group 1 comprised 15 cases which presented untreated acute gonococcal infections. Group 2 comprised 10 cases which were in the subacute or chronic stage and had been treated medically for gonococcal infections in the same clinic from one month to three years previously. It was difficult at the time of readmission to demonstrate the gonococcus among the mixed bacterial flora in a few instances, but by careful investigation and repeated examinations of the swabs taken

from the vaginal and cervical mucous membrane, the bacteriological department was able to demonstrate, "organisms typical in morphology and distribution to the gonococcus". The œstrogenic substance used was theelin.* The first group of acute cases received an average of 12,000 international units, as compared with an average dosage of 8,500 units in the second group. The duration of treatment averaged 56 days in the first instance and 34 days in the second. These figures include the duration of treatment for any recurrence or complication. According to the classification of Miller, as described above, the results of the present entire series were as follows:—(1) *Good*, (with no recurrence of discharge); 17 cases, or 68 per cent of the group. (2) *Temporarily controlled*, (recurrences within the first 6 months); 7 cases, or 28 per cent of the group. (3) *Failures*, (infection uninfluenced by treatment), 1 case, or 4 per cent of the group.

It was interesting to note that 6 of the 7 cases in which relapses occurred were found on vaginoscopic examination to have a chronic endocervicitis which proved to be a focus for the gonococcus. The duration of this complication varied from two to three weeks under active treatment, and once negative smears were obtained no further recurrences occurred.

In 50 per cent of the cases biopsies were taken of the vaginal mucosa at the termination of treatment, to determine what effect there might be from œstrogenic substances. I also obtained biopsies from normal children of similar age periods, and submitted them for serial section and comparison to the Pathological Department. Dr. F. W. Wiglesworth reported a definite change from the normal structure, with a distinct increase in the cell layers of the squamous epithelial type. I was also able to confirm the typical Lewis reaction in the vaginal smears during active treatment.

There are many aspects of the difficulties one encounters in the treatment of this condition. It is my impression, both from the group of cases reported and a much larger series observed over the past year, that one must eradicate every focus of the infecting organism before the patient is discharged if a permanent cure is to be hoped for. Therefore, the common sites, such as the cervix, urethra and rectum, should receive careful inspection and bacteriological examination at the termination of treatment. The cervix was found to be a focus in 24 per cent of the series presented. Bernstine[1] records the urethra as a focus in about 10 per cent, and believes the rectum to be a similar focus in a still higher percentage.

It is obvious that a large percentage of cases in the acute stage are complicated by infections of the urethra and rectum and are frequently undiagnosed. These conditions less frequently constitute a menace to the duration of a symptomatic cure. The infections involving the cervix do however present a more serious complication, and, it is my impression, are responsible for a large number of the recurrences reported.

The plan of treatment is to use large doses of the hormone having a slow rate of absorption over a period of six to seven weeks, in order to obtain a vigorous squamous-cell reaction of the vaginal mucosa. The method believed to bring about a maximum reaction with the least amount of medication is the tri-weekly injection of 1,000 international units each. The treatment should be continued until a symptomatic and bacteriological cure results. A routine vaginoscopic examination should be carried out in each case before discharge, to rule out the presence of cervical infection or adhesions of the vaginal mucosa. The local application of antiseptic solutions such as metaphen, (1-500), acriflavine, (2 per cent), or nitrate of silver (1 per cent), to the cervix, can best be accomplished with a small children's proctoscope, and in such manner should be carried out two or three times a week, until negative smears are obtained and the inflammation has subsided.

The matter of constitutional treatment should be considered of major importance, as there must be a natural tissue reaction as well to aid in the eradication of the local infection. The recognition and treatment of complications as they arise should receive prompt attention. It is agreed by most authorities that the administration of the hormone should be discontinued for a time in the face of symptoms which simulate tubal extension and pelvic peritonitis. Isolation of the child in the home must be made a special precaution, and the primary source of infection

* Supplied through the courtesy of Parke, Davis & Company.

should be ascertained and treatment attempted.

I feel that in a condition such as this, in which our medication is so inadequate, œstrogenic substances can be used to advantage, even though the results still leave something to be desired.

SUMMARY

A series of twenty-five cases of vulvo-vaginitis in children is reported.

The œstrogenic substance used was theelin.

The dosage varied from an average of 12,000 international units in the acute cases to an average of 8,500 in the mild types.

Symptomatic and bacteriological cures were obtained in a period averaging 45 days.

Recurrence of the discharge occurred in 28 per cent of cases; 24 per cent of this group were complicated by a focus of the infection in the cervical region.

Vaginoscopic examination should be adopted as a routine method at or near the termination of treatment.

The method of treatment found most satisfactory, was doses of 1,000 international units of theelin tri-weekly.

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CYSTS OF THE SEMILUNAR CARTILAGES OF THE KNEE JOINT

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NOTWITHSTANDING the probability of repetition it seems wise to contribute further to the rapidly accumulating mass of information regarding pathological changes in the knee joint, particularly in respect to cysts in or on the fibrocartilages. Since Ebner's[1] classical description in 1904 interest has been aroused, slowly at first but more recently gathering momentum, until now the diagnosis and treatment of cysts of the external fibrocartilage is quite an easy procedure in most cases. On the other hand, views on the pathogenesis become more conflicting with succeeding articles, and not until all the theories have been aired will a correct understanding be gained.

The writer's interest in the knee joint and its troubles has been constantly stimulated by Timbrell-Fisher's admirable book.[2] Having tried many of his observations and found them good, his wide knowledge is freely borrowed in this discussion.

It has been the author's fortune in the past few years to have had among his private patients 7 with cysts of the fibrocartilages of the knee joint. One of these, an internal cartilage with a large multilocular cyst, was associated with a complete oblique medial tear, following a rather severe injury to the right knee some months before the patient came under the writer's care. The diagnosis was made of a severely injured internal cartilage and the cyst was disclosed at operation. Of the other six cysts, two were found at operation for a tear of the lateral meniscus producing fairly clear-cut symptoms. Four cases presented visible, palpable cysts, the picture of which is now well established in the literature. In all, five cysts have been removed with complete alleviation of symptoms, while two remain in their respective hosts' knees, one possibly gaining a little in size and causing a little more trouble as time goes on. One man, a marine fireman, will probably require exploration of his knee-joint in the very near future, as he is having increasing difficulty in negotiating the iron ladders of his engine-room. The other, in a sedentary insurance dealer, who has no further interest in athletic pursuits, will probably remain where it is. His knee is giving little trouble.

All of the series occurred in males. Their ages, at the time of coming under observation, ranged from 20 to 34 years, only one being over

30. There was a definite history of injury in every case. The longest interval between injury and the time of coming under observation was nine years; the shortest between definite injury and operation was six weeks. This last, a very recent case, afforded the rare privilege of observation from the day following injury. (Case 5).

These observations lead the writer to the *post hoc ergo propter hoc* conclusion that cyst formation in semilunar cartilages is a direct result of trauma of some type, and that it may be mild or severe. This view is put forth in the most recent article to hand on the subject, that of Taylor,[3] who brings up to date a statistical study of the literature. It is difficult, however, to subscribe to the direct contusion theory as opposed to one of strain. The knee joint is constantly liable to rotation strain, whereas it is difficult to conceive of a blow so nicely adjusted as to strike directly the peripheral margin of a semilunar cartilage. It has been shown that the semilunar cartilages have a considerable blood supply in the peripheral portions, and this is where the cysts occur, particularly in the mid portion, the most highly vascular of all. Cysts have not been described in the thin, inner half of the cartilage, which, presumably, depends for its nourishment on the synovial fluid and is adjusted to that. Is it not, then a direct and logical explanation of cyst formation to consider that it follows vascular obliteration in the most highly vascular portion of the cartilage, the result of injury, be it strain or a direct blow? The pathological changes that then take place are clearly set forth in Taylor's excellent article noted above.

CASE 1

T.S., a male, aged 27, was first seen in July, 1935. In the summer of 1934 he was drilling on a jack-hammer when the steel broke and he fell 15 feet, landing on his feet. His right leg was jammed between two stones and was wrenched. The knee became very painful and swelled immediately. When the swelling had subsided under rest and heat the knee would "jam" almost daily, and he would have to get someone to straighten it. In March, 1935, he was re-admitted to the hospital with "something protruding" from the inner side of the joint. This subsided but the locking continued. On examination there was slight fluid in the joint, with the greatest tenderness over the anterior end of the internal semilunar, increasing on extension. There was also lateral joint-line tenderness, and he stated that recently his pain had been more in the lateral side of the joint. Extension was limited; there was no lateral instability, but there seemed to be slight antero-posterior instability in extension. Operation disclosed a complete oblique medial tear of the internal meniscus, with the anterior fragment almost free in the joint, and a large typical multilocular cyst in the periphery of the anterior end of the posterior fragment. Convalescence was uneventful, except for some synovitis which ultimately cleared up, and at the time of writing the man has a normal knee.

CASE 2

E.R., a male, aged 20, was first seen in May, 1934. He stated that in 1930 he twisted his right knee while running a mile race. In a month his knee felt better, but later, in another race, it was greatly aggravated. Later on a swelling was noted over the lateral joint line which nearly disappeared on flexion and extension but was most prominent in semi-flexion. When he walked he felt something "catching" in his knee. The knee would ache when he first went to bed. Examination showed a typical cyst of the external semilunar cartilage. The whole cartilage was removed intact with a large multilocular cyst attached. A partial medial tear of the cartilage was noted. Recovery was uneventful; he was walking in ten days and there has been no recurrence.

CASE 3

E.A.Mc., a male, aged 23, was first seen in October, 1935. When playing rugby at the age of 14 his knee was "put out" when he was tackled, and ever since he had considered it a "trick knee". On the slightest provocation, and even sometimes in bed it would "go out of joint". Examination showed little, the only physical finding being tenderness over the anterior attachment of the internal semilunar, increasing on extension. Extension was possibly slightly limited, the "screw home" movement being absent. A diagnosis was made of a loose fragment of internal semilunar cartilage, probably pedunculated, in the knee joint. At operation an old anterior tear of the internal semilunar cartilage was found and a bean-shaped pedunculated fragment was removed. On looking across the joint from the medial incision, a cyst the size of a filbert was seen at the periphery of the lateral meniscus. This was not visible from the outside. When the medial incision was closed a lateral pocket flat incision was made through which was removed the complete lateral cartilage, to which was attached a large multilocular cyst. No tear in the lateral cartilage was noted. Recovery was uneventful and the patient has remained symptom-free.

CASE 4

G.A., a male, aged 21, was first seen in May, 1936. In September, 1935, he had slipped on some loose bark on a log and twisted his left knee. The mechanism was not elucidated. He reported to the first-aid man and carried on. Later the same day he again twisted the same knee when he stepped on a rock. From then on whenever he would step on anything uneven he would have a violent pain in the left knee which would cease in a moment or two. In November, 1935, he suffered a fracture of the left index finger, and in April, 1936, a rather severe scalp wound. At this time he decided to give up logging and came to town to see about his knee. Examination showed a typical fluctuant swelling over the centre of the lateral joint line, most marked in semi-flexion. Complete extension was painful. At operation the usual multilocular cyst was found attached to the external semilunar, encroaching on the cartilage substance, but within the capsule. An incomplete medial tear was very evident. The whole cartilage was removed with the attached cyst, and at this time he is convalescing normally.

CASE 5

F.B., a male, aged 22. In June, 1936, he fell ten feet from a pile of lumber and twisted his right knee. He was unable to describe the mechanism of the twist but is sure that the knee was not directly struck. When seen the following day extension and flexion were both limited and painful. There was some fluid in the joint.

There was no instability, but adduction and abduction produced pain. The only point of tenderness, and it was decidedly acute, was over the joint line immediately between the popliteus tendon and the external lateral ligament. Internal rotation of the flexed tibia on the femur produced pain in the lateral part of the joint. It was considered that the evidence of an injury to the lateral meniscus was fairly definite, but one hesitated to explore. A month later a vague elastic swelling was barely discernible at the joint level behind the external lateral ligament. The fluid in the joint had disappeared and the range of motion had increased. The extremes of extension and flexion, however, were still painful and the tender point noted above remained decidedly acute. Two weeks later the joint was opened, using Timbrell-Fisher's pocket flap approach, and there was found a complete medial tear of the lateral cartilage and in its periphery, anterior to the tear, three tiny cysts of typical appearance, each the size of a black-headed pin. His convalescence was uneventful and he returned to industry six weeks after operation with an apparently normal knee.

CASE 6

A.C.B., a male, aged 24, was first seen in October, 1929. A year before, his knee was put out while playing rugger. After a short time he was symptom-free. Then gradually a swelling appeared over the outer side of the knee-joint, which remained stationary for some time. This cystic mass presented the typical picture of a cyst of an external semilunar cartilage, (beautifully illustrated in a photograph in Timbrell-Fisher's book), oval in shape and directed slightly obliquely behind and beside the external lateral ligament. It was most obvious on semiflexion. He occasionally has trouble in complete extension, but his physical life is very inactive and he declines to have the cartilage removed. Incidentally, he has nursed an ununited left carpal scaphoid for several years.

CASE 7

G.D.B., a male, aged 34, was first seen in July, 1936. While working in a crouched position in his engine-room some six months previously his right foot slipped on some oil on the iron grating and his foot shot sideways, twisting his knee. From then on he has had trouble with it of a more or less minor nature, but recently he has had increasing difficulty and pain in ascending and descending stairs and ladders. Examination shows again the typical elastic cyst over the lateral side of the knee joint, nearly the size and shape of an almond. It is moderately tender and almost disappears on extension and right-angle flexion, but is very prominent in semiflexion. Removal is anticipated at an early date.

To Mr. Taylor's statistical table can then be added the following: no. of cases, 7; sex, male; average age, 23; trauma, definite 7; cartilage affected, internal 1, external 6.

In conclusion, it may be accepted that in most cases the diagnosis of cyst of the external semilunar fibrocartilage is quite easy, for the reason that they are usually visible and palpable from the outside. In one case, although a large cyst was present, nothing could be seen or felt externally. There is insufficient material at hand to lay out any clear-cut indications of cyst of the internal semilunar fibrocartilage.

The writer believes that trauma, the exact nature of which is not yet understood, is the originating factor in cyst formation. Whether cysts are formed in the substance of the cartilage and extrude, or are formed in the periphery of the cartilage and intrude as well as extrude, cannot yet be proved. The theory of peripheral vascular obliteration is advanced as the causative pathological factor. The consensus, so far as treatment is concerned, is that the whole cartilage with attached cyst should be removed. This is easily accomplished through the Fisher[2] modification of the pocket-flap incision.

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AN ADDITIONAL NOTE ON THE INNERVATION OF TUMOURS*

BY HORST OERTEL

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SINCE my previous publications on tumour and cancer innervation a number of contributions dealing with the same and cognate subjects have appeared, which, on account of their importance, it may not be amiss to bring to wider attention.

A monographic article by M. Athias and M. T. Furtado Dias[1] represents an additional careful study into innervation in experimental animals and in man. Their results are in general quite confirmatory of Itchikawa's and our findings[4] as regards the presence of tumour nerves in animals (mouse and rat), and in the human being. Subsequently, several other noteworthy contributions have been published, which, although not dealing with tumour innervation directly, closely touch upon it.

The first of these concerns microscopic observations of the nervous apparatus of the stomach in peptic ulcer by Philipp Stöhr.[7] Stöhr has succeeded in demonstrating, as far as I know for the first time, the finer nerve changes

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in and around gastric ulcers. He found extensive processes of nerve increases in Auerbach's and Meissner's plexus, with considerable augmentation in the axis cylinders and Schwann's cells, and in two cases a neurinoma in Meissner's plexus close to the ulcer. Lymphoid cell infiltrations were also found in these nerve plexuses. The ganglion cells were much changed, with neurofibrillar outflow and hydrops; they shrink finally to destruction. Nerve fibres bud into the ulcer with degenerative and regenerative processes, and a similar invasion of the nervous bundles into the ulcer granulation tissue and scar occurs. Indeed, the whole ulcer may be traversed in all its layers by strong nerve bundles towards the surface. These newly formed nerve bundles exhibit a characteristic spiral course of their axis cylinders. They grow with the granulating tissue and are finally lost in hypertrophic (!) glandular masses. Stöhr points out that this growth of new axis cylinders into the granulating and necrotic zones of the ulcer base and fibrous edges resembles the axis-cylinder formation and manner of growth which I have described in cancerous tumours—an important and suggestive finding.* In one very instructive instance in which a stomach was removed for ulcer symptoms, but no ulcer or other anatomical gastric lesions (gastritis) were found in well over 1,000 sections, ganglion-cell changes were discovered in quite different regions of the stomach. Stöhr regards the sympathetic nervous system as syncytium (no neurones; therefore, without entities consisting of ganglion cells, neurites and dendrites). He draws renewed attention to its vegetative importance, and holds that the intramural gastric nervous apparatus is really a single, plasmatically connected syncytial system. As a consequence of this, it is erroneous to speak here of diseased ganglion cells and nerve fibres, but these changes represent the anatomical expressions of a totally involved intramural nervous apparatus of the stomach.

If this striking contribution deserves wider attention, more particularly in the possible relation to the origin and fate of these ulcers there are several others which also demand consideration. Scherer[5] has made observations which strongly point to a causal connection of nerves and nerve lesions with abnormal growth. He studied a case of neurofibromatosis with circumscribed giant growth, particularly of the muscular coat of the œsophagus, stomach, and appendix. In the case of the appendix there was a massive new growth of the submucous nervous tissue; in that of the stomach and œsophagus a tremendous, practically pure, hypertrophy of the muscle, with a corresponding quantitative increase of the nervous elements. There existed also a strong neurinomatous change in both vagi, and to a lesser extent in the sympathetic. Scherer showed that these neurofibromatous thickenings had not led to any injury or destruction of nerve fibres, so that an interference with their function apparently did not exist. He concludes his extensive study as follows: "Experimentally a growth-increase of the stomach has been found after ligation of the vagus. The vagus is the motor nerve at least of the upper part of the digestive tract. These facts seem to indicate that in the instance here described, in which the vagus showed a strong neurinomatous change, the elective hypertrophy of the motor apparatus of the œsophagus and stomach is causally connected to a chronic vagus irritation." Scherer holds that the genesis of the giant growth of muscular tissue in œsophagus and stomach permits the assumption of a trophic influence. His case, therefore, furnishes the first instance in human pathology in support of the results of animal experiments by Timme[8] on the nervous mechanism in the production of hyperplasia.

A further reference should be made to an article by Stalmann,[6] which also deals with the relationship of neurofibromatosis, more particularly to skin and bone changes. He comes to the conclusion that there is a genetic relation between nerve changes, pigmentation and skin tumours. In the bony system, also, reconstructive changes with loss of bone and new bone formation, loss of calcium and increase of calcium, were noted. Thus, all sorts of skeletal irregularities developed. Stalmann considers that the neurofibromatosis has its origin in the embryonic-developmental period and represents

*"Particularly clear illustrations have been furnished by Oertel, who was able to demonstrate in cancerous tumours plexus and network formations of the advancing axis-cylinders, naturally deviating from the normal. I have frequently seen such pictures in the granulating district and in the necrotic zone of the ulcer base." (Stöhr l.c., p. 617). Stöhr reproduces an illustration of axis-cylinder fibrils in intimate contact with a fibrocyte, much as I have seen it in tumours.

a blastomatous lesion of the nervous system. In this developmental pathological process, abnormal, morbid irritations arise and are conveyed by way of the nervous system to the organs of the body. These organs are then functionally and anatomically altered. I will refrain here from a discussion and bearing of these findings, more particularly in the relation which the nervous system probably has to the normal or abnormal structural tissue balance in its "morphological fluidity" and age period changes, a matter with which I have repeatedly dealt in previous publications.

Finally, J. Christ,[3] in a very thoughtful and detailed study, has extended the important earlier observations of Cheatle[2] on the regionary extensions of facial cancers to a coherent trophic nervous organization attached to the sensory system and distinguishable into nervous territories. This pluri-innervation is, in its terminal districts, causally concerned in the different normal and abnormal changes in the skin, for example, in the dorsal line and mane and the polygonal skin pattern of certain vertebrates. In man they permit us to recognize a relation to circular, vertex baldness, rodent ulcer (in the face, the orbital field), and of cancer of the lower lip.

The principle which I enunciated as a result of previous investigations, that tissues never move in their parts but only as indivisible entities, is clearly strengthened by the findings here recorded. But these observations are not only directly or indirectly contributory to our results in the finding of nerves in tumours; they definitely raise the question of the causal relations which irritative or other nervous factors may have in the production of pathological growths, including tumours. I trust, therefore, that this brief account will draw renewed attention to this possibility and stimulate further investigation of this issue. It is much to be hoped that the improvements in histological technique and a further constant application to these problems by those well prepared and trained for that pursuit will lead us to further demonstrable facts, which, I believe, will ultimately be of the greatest service in obtaining an anatomically better founded comprehension of tumour growth.

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Case Reports

AN UNUSUAL INTRATHORACIC TUMOUR

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Intrathoracic tumours are noted for a wide range of symptomatology, and for this reason are often overlooked until far advanced. The case herein presented exhibited an unusual set of symptoms and is therefore considered worthy of record. Although the tumour type is not uncommon it is the belief of the writer that this particular site for the growth is rather rare.

B.S., male, aged 35, a farm labourer.

Complaints.—(1) A dry, cracked right hand; (2) inability to perspire on the right side of the face, right arm and right hand; (3) pain in the right arm and hand.

History of illness.—The patient stated that he was perfectly well until five years before, at which time he began to have pains in the right elbow and right forearm. These pains were severe and of a steady aching nature, making him generally miserable and interfering considerably with sleep. A doctor was consulted who treated him for "rheumatism" without good result. The severity of these pains had lessened, but they had never entirely disappeared and at the time of consultation were still a very troublesome feature. Soon after the onset of pain he noted an increasing dryness of the right side of his face, right arm and right hand, and finally perspiration in these parts entirely disappeared, even on the hottest day. With this the skin on the palm of the hand became very thick and calloused and, of course, extremely dry. The skin on the palm cracked

easily and left deep bleeding furrows. The hand was constantly sore and interfered greatly with his work. During this time friends noted a changed appearance of the right eye, that is, an apparent drop of the lid and a narrowing of the pupil. He, however, complained of no ocular symptoms. Considering his occupation, the above-named symptoms were an extreme handicap, to say nothing of the constant nagging pain in the arm, and for the past five years he had travelled from doctor to doctor in search of relief. An x-ray of the chest was never taken. The personal and family histories were irrelevant.

On examination the patient was found to have a right Horner's syndrome. The pupil was small but reacted to light and accommodation. The palpebral fissure was markedly narrowed. The right eye was considerably drier than its mate. There was an "apparent" ptosis, but the lid was easily elevated by looking upward. The day he visited the office was warm. The right side of his face was perfectly dry. This included the forehead. The left side was moist and perspiring freely. The right jugular vein was full; the left appeared normal. No tumour mass was felt in the neck, but there was a definite bruit over the right subclavian artery and a feeling of something solid below it. The right axilla was perspiring slightly, but the right arm and hand were dry. The left was moist and soft. The skin on the palm of the right hand was very thick, dry and cracked.

There was a band of anæsthesia about ½ inch to 1 inch wide over the posterior surface of the ulna. This extended from the elbow to the wrist but did not come around in front (T 1). No other areas of anæsthesia were made out. The strength of the muscles was equal in both arms in all groups. Blood pressure right 130/80; left 120/90.

General physical examination revealed nothing of note. Examination of the chest showed a small area of dullness above the 1st rib, but other than this no abnormality was made out.

Preliminary fluoroscopic examination showed a rounded tumour situated in the dome of the right chest. This was non-pulsating.

The routine laboratory examinations revealed nothing of note. The red blood cells numbered 5,080,000; hæmoglobin 85 per cent (s); white blood cells, 10,000; polymorphonuclears, 68 per cent; lymphocytes, 22 per cent; transitionals, 5 per cent; eosinophiles, 1 per cent; basophiles, 3 per cent. The blood Wassermann test was negative. Pneumothorax, for the purpose of diagnosis and mediastinal stabilization, was initiated on April 6, 1936. Refills of 450 or 500 c.c. of air were made every second or third day until April 18, 1936. At this time there was a 60 per cent collapse of the lung.

Skiagrams, taken on admission to hospital and during the pneumothorax period, were reported on by Dr. W. H. Eagar as follows, April 9, 1936.

"*Right side.*—Circumscribed mass size of a small orange lying at a level of 1st to 4th ribs posteriorly, left margin to median line of vertebræ. Partial pneumothorax extending down to 4th rib laterally. The upper lobe is compressed and shows the indentation of the tumour with thickened pleura (see Fig. 1). Interlobar pleura thickened—central—at level of 3rd rib. Thickening of hilus with moderate peribronchial thickening.

"The *left lung* shows moderate general peribronchial thickening. No pathological focus in either lung or evidence of metastases."

On April 10, 1936, a thick barium meal was given. There was an arrest of the meal at the aortic knob. The œsophagus was displaced to the left and flattened by the tumour mass. The medial border of the tumour was reported as lying behind the trachea. Further x-rays were taken which need not be reported.

On April 18, 1936, a thoracoscopic examination was carried out. The thoracoscope was introduced into the 3rd space in the anterior axillary line and the interior of the chest observed. There was approximately a 60 per cent collapse of the lung (uniform). The apex was adherent by a curtain adhesion to the dome on the mediastinal side. The lung appeared normal in every way. The parietal and visceral pleuræ were thin and glistening. There was no fluid in the pleural cavity. The right auricle was seen pulsating in a free pericardial sac. No mediastinal bulging was seen. The phrenic and vagus nerves were seen descending on the side of the mediastinum. The internal mammary vessels were easily visible. The tumour was readily visualized. It was situated in the curve of the first rib and filled the whole dome, lying directly on the neck of the first rib and following its curve laterally. The pleura over it was thin and glistening, and did not appear adherent or infiltrated. Running over it, and in front of it, were the subclavian vessels and brachial plexus. These

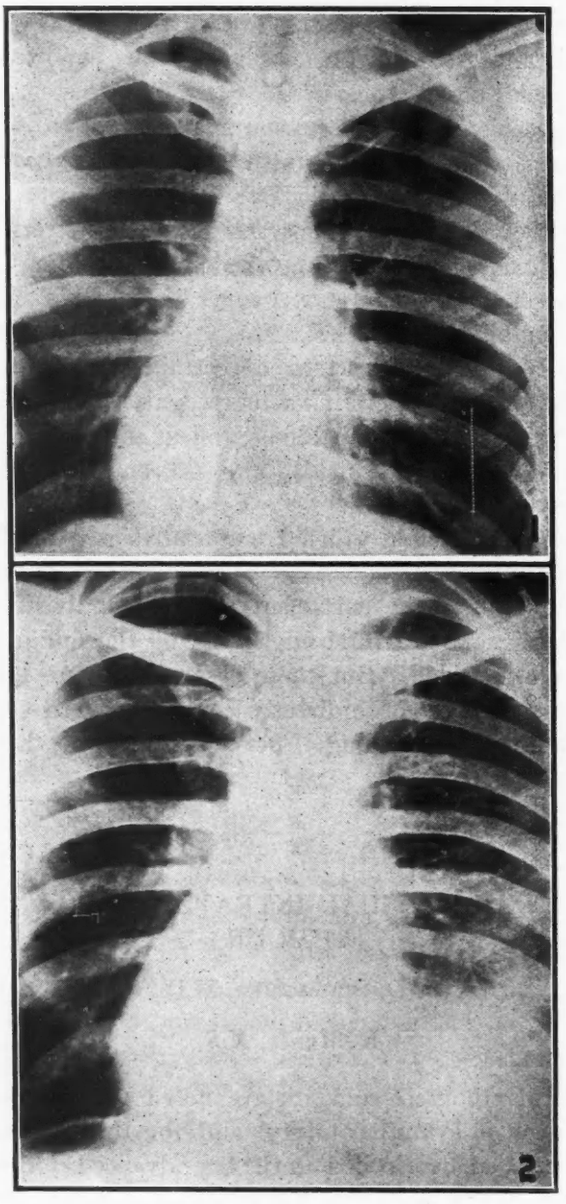

Fig. 1.—Partial pneumothorax. A smooth round tumour in the dome of the right chest. Apex of lung indented by tumour mass. **Fig. 2.**—Two weeks after operation. Absence of first rib and anterior half of the second. Diaphragm high. Complete expansion of the lung.

structures were very closely applied to the anterior aspect of the tumour. The thoracoscope was removed, the skin clipped and pressure dressings applied. On April 22, 1936, under avertin and gas anæsthesia, the tumour was removed.

Operative technique.—An incision was made from the tip of the coracoid process to the manubrium sterni, parallel to and about 2 inches below the clavicle and then curved downward laterally to the sternum.

The pectoralis major was split between its clavicular and sternal attachments and retracted. A portion of the sternal attachment had to be divided for adequate exposure. The muscles were stripped from the chest wall and the upper ribs exposed. The long thoracic nerve was freed and retracted laterally. The first rib with its cartilage was resected subperiosteally. The tumour was then easily felt lying under the bed of the rib. The subclavian vessels and brachial plexus passed directly over it in front. An attempt was made to free the growth extrapleurally, but it was too deeply and too firmly embedded to allow of this. An anterior portion of the 2nd rib and its cartilage were then removed subperiosteally. Under positive pressure anæsthesia the pleura was then entered rather widely. (NOTE: There was no change in respiratory or pulse rate and no mediastinal flap). The tumour was now well exposed. The pleura on its inferior surface was incised and peeled easily. The tumour was then enucleated carefully with the finger, as one would a prostate. The posterior part lay deep in the vertebral gutter; medially, it lay behind the trachea and directly against the œsophagus. Care had to be exercised to avoid tearing into the œsophagus. The growth was then completely freed and came out clean, leaving no part behind. There was very little oozing from the tumour bed. The opening in the pleura was repaired, the lung being re-expanded under positive pressure before final closure was made. The wound was closed. The condition of the patient at the end of the operation was excellent.

Gross appearance of the tumour.—It was roughly the shape of a curling stone, rounded, but flattened. In the fresh state it was about 3 inches in diameter and about 2 inches thick. It was completely encapsulated by a dense fibrous capsule and at no place was there any evidence of infiltration. It was hard, but smooth. No nodules were seen or felt.

It was soft in consistency and cut easily. The cut section was a mottled yellow with one central necrotic spot. The cut tumour bulged from its capsule. Dr. Ralph Smith, pathologist, reports the following histological examination. "The tumour is composed chiefly of long thin spindle cells which are arranged in whorls and fasciculi. In many areas the cells have undergone hyaline degeneration and necrosis. Scattered throughout the tumour are other cells which are undoubtedly producing nerve fibrils. The tumour appears to be well differentiated and mitotic figures are extremely rare. It appears to arise from the sheath of Schwann and is therefore a neurofibroma or neurinoma. There is no evidence of malignant transformation."

An x-ray taken on April 26th (four days after operation) showed the lung to be practically re-expanded and there was no fluid in the chest. The right diaphragm was high, indicating phrenic nerve damage. This point was later proved by fluoroscopic examination. The diaphragm was high, moved paradoxically, and there was a positive Hitzenburger. Convalescence was entirely uneventful. The patient was allowed out of bed on the 12th day after operation and discharged five days later. The x-ray on discharge is seen in Fig. 2—the lung is re-expanded and the lung fields are clear. The diaphragm is still high.

The operation has resulted in complete relief of the pain. The skin of the palmar surface of the hand has thinned out and now feels like that of the opposite hand. It is soft and has lost entirely the horny feel it previously had. There is a degree of moisture, and, although the hand does not perspire freely, there is definite sweating between the fingers and at a small spot, the size of a quarter, proximal to the index finger. The area of anæsthesia on the back of the forearm is still present. The Horner's syndrome, although still present, is decidedly less marked than previously. A recent fluoroscopic examination shows complete return of function of the diaphragm. It has regained its normal position and moves synchronously with its mate.

A point of discussion arises as to what mode of approach to such tumours is best. In this particular case a posterior thoracoplasty incision, with subperiosteal resection of the 1st and 2nd ribs, would probably have allowed of extrapleural removal, thus doing away with the danger of large, open pneumothorax. It was felt, however, that should the tumour be adherent to the sheaths of the great vessels, the brachial plexus, or mediastinal structures, that these could best be protected through the anterior approach. The danger of large, open pneumothorax is also minimized by preliminary pneumothorax treatment and the use of positive pressure anæsthesia.

CANCER OF THE ŒSOPHAGUS: APPARENT GOOD HEALTH THREE YEARS AFTER X-RAY THERAPY

BY GEO. H. MALCOLMSON, M.D. AND P. H. MALCOLMSON, M.D.

Edmonton

Mrs. L., a white female, aged 43 (Royal Alexandra Hospital No. 45866) was admitted December 8, 1933, complaining of difficulty and pain on swallowing since August, 1933.

Present illness.—The patient at first noticed some difficulty in swallowing solids. This gradually became worse until she could only swallow fluids with a great deal of difficulty. She had become very weak and her weight had decreased from 161 to 87 pounds in two months.

Examination.—A pale, emaciated female, who on attempting to swallow made a gurgling sound and regurgitated her food. Hæmoglobin 50 per cent; red blood cells 3,100,000; white blood cells 6,000. Wassermann test was negative.

X-ray examination with a thin suspension of barium in water showed almost complete obstruction of the œsophagus at the level of the body of the 6th cervical vertebra (Fig. 1). *Diagnosis:* probable carcinoma.

Œsophagoscopy.—December 11, 1933. A hard, smooth constriction was noted posterior to the cricoid cartilage. *Biopsy:* pathological report—epithelioma of the œsophagus.

On December 29, 1933, protracted fractional x-ray therapy was started. The technique was as follows: 200 Kv., 5 ma., F.S.D. 55 cm.; filter 1½ cu. and 2 al.; 2 lateral ports 10 x 15 cm. Daily dose, 150 r. (measured on skin).

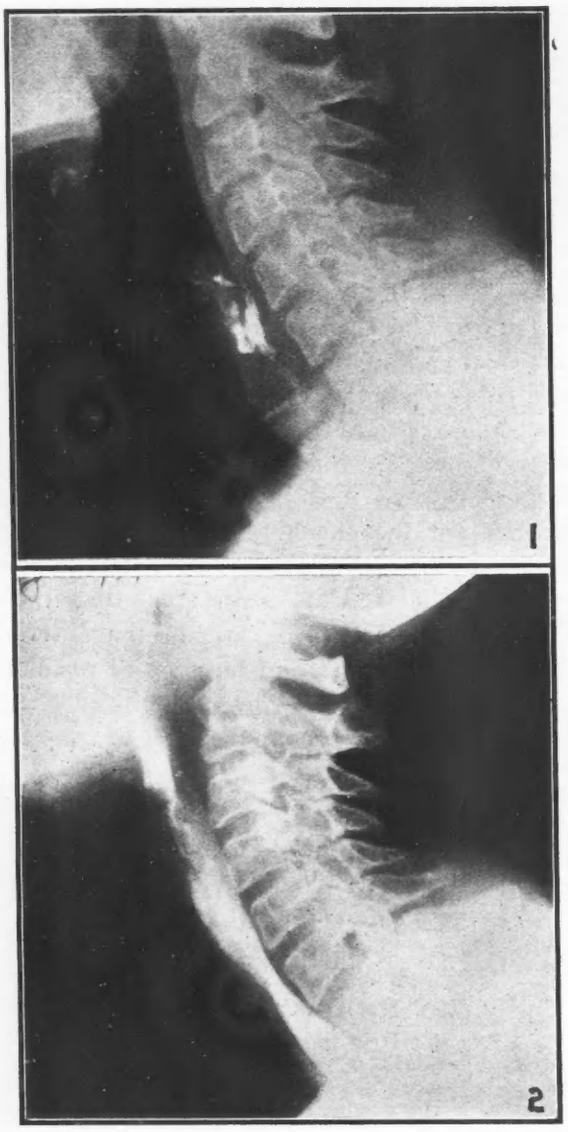

Fig. 1.—Before treatment. Almost complete obstruction of the œsophagus at the level of the body of the 6th cervical. (Note displacement of the trachea anteriorly by the tumour mass.) **Fig. 2.**—Four months after treatment. Only a slight smooth constriction at the site of the tumour growth. (Note that bulging of the trachea seen in Fig. 1 has disappeared.)

This treatment was given daily from December 29th to February 15th. From the first week after beginning it the condition of the patient improved. She was able to take fluids easily and was beginning to handle solid food when radiation epidermitis began. Because of early skin reaction treatment was discontinued when a dose of 3,750 r left and 3,300 r right side of the neck had been given.

The patient's condition continued to improve, and she was discharged February 28, 1934. At this time she was able to eat any type of food and had gained in weight from 87 to 105 pounds.

X-ray examination February 28, 1934, showed a slight constriction at the level of the 6th cervical body, but a free passage of barium into the stomach.

Œsophagoscopy.—A post-cricoid cicatricial contraction, but no evidence of tumour growth.

Fig. 2: The patient was re-admitted on April 11, 1934. X-ray examination and œsophagoscopy showed no evidence of growth. However another course of treatment was thought advisable since the patient's skin had proved too sensitive to complete the original course; 2,400 r units were given to each side of the neck.

In conclusion, I might say that the pathological sections have since been reviewed and would probably grade 1 or 2, Broders, and yet were apparently very radio-sensitive.

A CASE OF HYPOTHYROIDISM

By H. E. Macdermot

Montreal

The benefits usually produced by the proper treatment of hypothyroidism never seem to lose their impressive character. In Osler's incisive words:

"The results, as a rule, are unparalleled by anything in the whole range of curative measures. Within six weeks a poor, feeble-minded, toad-like caricature of humanity may be restored to mental and bodily health."

The case I am reporting, whilst lacking quite such highly dramatic elements, is yet sufficiently satisfactory, and also presents a point of interest in connection with the treatment.

The patient, aged 55, a doorman, was admitted to the Montreal General Hospital complaining of great weakness and dyspnœa on slight exertion, swelling of the hands, aching legs, and loss of hair. These symptoms had become progressively worse within the last four years.

The only significant point in his family history was that two of his sisters were suffering from similar complaints.

His personal history showed no evidence of previous cardiac or pulmonary disease. He took some alcohol, and smoked in moderation. His wife, who died of tuberculosis some years ago, had had two miscarriages. There were two healthy living children.

The patient had been able to work until about a month before admission, when he had developed severe "lumbago". He had had to keep to his bed for a week, and had lost 18 pounds in four weeks. His speech also had become noticeably thickened and deep in recent months, and his limbs got cold very easily. He saw a physician, who told him that he was anæmic. Although no basal metabolic test was done then, hypothyroidism also was evidently suspected, and the patient was put on large doses of thyroid extract, as much as 15 grains a day. He found himself getting weaker and very "jumpy" and was referred to the hospital.

His condition on admission was immediately suggestive of hypothyroidism. The complexion was of a muddy, yellowish colour, and his features were pudgy and coarse. His skin in places was thick and rough, and the hands were thickened without being œdematous. He had lost most of the hair of his head, only a few strands being left on the crown. The axillæ were quite smooth, but there were a few hairs on the chest, and the usual pubic growth. His voice was low and thick, and his mental reactions rather slow, although quite clear.

The physical examination otherwise contained little of note. The temperature, pulse and respirations were normal. The heart and lungs were clear, and the abdomen was negative. The blood pressure was 128/24. There was some loss of vibration sense, but no other sensory disturbances. At first he complained of a great

deal of pain in the submaxillary region, and there was much tenderness there, but no glands could be felt, and no explanation of the pain was found.

Hæmatological examinations showed a moderate secondary anæmia, with blood cells of normal size and shape. The stomach contained a normal amount of acid. The electrocardiogram was normal. The blood chemistry was normal, and the blood Wassermann reaction was negative. The urine was negative.

The basal metabolic test naturally was turned to, with every expectation of its supporting the diagnosis of deficient thyroid secretion, but it was rather disconcerting to find that the reading was plus 3. It was felt, however, that the heavy thyroid dosage in the previous month might be responsible for raising the rate, and the test was repeated in four days, when it was found to be minus 3. A third test was done four days later and this time the result was minus 13. Along with this, the pulse rate showed a distinct slowing, from an admission rate of 74 to 62, and there was a gain of over three pounds in body weight. Even before this, however, the diagnosis of hypothyroidism seemed unavoidable, and the man was put on thyroid extract, receiving two grains of the dried gland twice daily, along with a high iron diet. Improvement began at once. His speech became clearer, he lost his limb pains, and felt able and desirous to move about. Within two weeks of admission he was well enough to go home, and was back at work a few days later. His hair has reappeared on his head, and his employers comment strongly on his improved capacities. He is on a maintenance dose of a grain of thyroid extract twice a day.

The case illustrates the danger of excessive thyroid medication, even in the presence of definite hypothyroidism.

BRAIN ABSCESS: RECOVERY

By J. H. DUNCAN, M.A., M.B.

Sault Ste. Marie

In September, 1935, a young man of 21 years, was taken ill with tonsillitis, followed by left-sided otitis media and mastoiditis. He was admitted to the General Hospital on October 2nd under Dr. A. R. Stevenson. A simple mastoid operation was performed the next day, much necrotic bone being removed and good drainage established. Thereupon his temperature began to fall from 103° F. on the day of admission, to reach 98.6° F. on the eighth day. That same evening, however, he had chills, headache and vomiting, and his temperature returned to 103° F. Under general therapeutic measures he seemed to make progress in the next two weeks but was never quite free from headache for any length of time. On the last day of October he was allowed up in a wheel chair for half an hour, and that night his headache was worse; he vomited and began to be drowsy. By November 3rd he was comatose. His temperature ran from 99 to 100° F., his pulse and respiratory rate being both normal.

Dr. Stevenson being out of town, the writer was asked to see the patient. He could not be roused, his left pupil was dilated, the right normal; the fundi were both normal. His blood pressure was 170 systolic. His spinal fluid contained 22 polymorphonuclear leucocytes per c.mm. It seemed clear that this young man was now suffering from an abscess in the brain, secondary to his ear infection. The problem was to locate the abscess and establish drainage. The difficulties and dangers of interference were explained fully to the near relatives as well as the consequences of non-interference, and they urged us to do all we could to save his life. Several members of the hospital staff had seen him and all agreed on the diagnosis. Drs. McRae and McCaig were called in to assist, and at 5 p.m. on November 3rd a temporo-parietal flap was reflected and a trephine opening made in the skull just above the left ear and three-quarters of an inch above the external meatus. While novocaine had been injected, his comatose condition rendered this almost superfluous. When the dura was incised a long needle attached to a 20 c.c. syringe was used to explore for the abscess. It was first directed back to the cerebellum, piercing the tentorium, but nothing was found. Then it was turned forward toward the temporal lobe, and at a depth of about two inches the needle entered an abscess cavity, and about 40 c.c. of flocculent yellow pus were withdrawn. A soft rubber drain was pushed down to the cavity and the outer end sutured to the edge of a stab wound made in the skin flap. The latter was then replaced and sutured with interrupted silk-worm gut. Moist bichloride gauze dressings were applied. The next morning the patient wakened and tried to talk, and each day saw some improvement, although there was a slowness in movement of the right hand and considerable aphasia. The wound did not discharge very freely, and at the end of a week the patient began to complain of his head again, vomited, refused his food, and a swelling appeared over the trephine opening. Once more he sank into coma. Without any anæsthetic the original flap was reflected and a firm clot and some cerebral tissue were found blocking the opening. This was cleared away and a small catheter inserted for drainage. The purulent discharge after this was increased in amount, and for a few days he had fever, very rapid pulse, and lack of bladder control. About November 17th his convalescence really began. All drainage tubes were out by the 20th, and from then on his recovery was uninterrupted. The patient was presented at a staff meeting on the day of his discharge, December 11th, at which time he was walking about free of all symptoms except auditory aphasia. He could hear the word, but could not think of the meaning, although he repeated it over and over. He went home to the country before New Year's day and did not return until June, 1936. At that time he was looking the picture of health, talked very well, answering all questions quickly. No speech defect was noted, although he stated that he had some difficulty with certain words. However, this cleared up by September, just a year from the beginning of his illness.

As far as I have been able to ascertain this is the first case of brain abscess successfully operated on in either of the hospitals in this city. In fact, it is the only case in the hospital records in which the diagnosis was made and verified ante mortem. Cushing states in the article which he contributed to Keen's Surgery that abscesses of the brain secondary to ear-infections are found either in the temporal lobe or in the cerebellum, more commonly in the former. He also states that without operation the outlook is absolutely bad, and that with operation, even in experienced hands, the mortality is 50 per cent. We feel therefore that we are singularly fortunate in the favourable outcome of this case.

Clinical and Laboratory Notes

RENAL FUNCTION BY THOMAS' INDEX OF ELIMINATION

BY EARLE R. HALL, M.D.

Vancouver

For many years surgeons dealing with prostatic operative procedures have been confronted with the problem of renal function. It is well known that patients with prostatism usually show some degree of diminished kidney function—more so in those carrying on over longer periods of time before seeking relief, and especially so where infection has invaded the urinary tract. Before prostatic surgery is carried out, whether by open methods or one of the now more widely adopted closed methods, the kidney action of these patients must be maintained at a certain point of stability or disaster is very likely to follow.

For many years various dyes known to be eliminated by the kidneys have been used as a method of arriving at the functional status of these organs. Several dyes have been proposed for this purpose, notably, fuchsin, methylene blue, rosanilin, indigo-carmin and phenolsulphonphthalein, but only the two last named have withstood the test of time and been generally adopted. As stated by Maurer,[1] their use has become more widespread and their usefulness has become more firmly established, until they form at the present time one of the most valuable aids in the diagnosis, prognosis and treatment of renal effections. Especially is this true in lesions of a surgical nature, whether these involve the kidneys directly or whether the kidneys suffer indirectly, as in the obstructive conditions of the lower genito-urinary tract, for it is here that the extra burden on the kidneys entailed in operative measures makes knowledge of the functional capacity a matter of paramount importance. Thus it is the urologist who is particularly concerned with this dye test, and his interest is directed towards a determination of the combined or total kidney function, and determination of the function of separate kidneys.

In this connection I wish to elaborate on the determination of the Index of Elimination of B. A. Thomas.[2] This index is the quotient obtained by dividing the percentage of dye excreted in the first period by that excreted in the third period—found to be 5.1 as normal when intramuscular injection of dye is given and hourly collections are obtained. To determine what collection periods were necessary for intravenous injections of dyes, Thomas found for indigo-carmin twenty minutes and for phenolsulphonphthalein fifteen minutes. For practical purposes three twenty-minute periods of collection were also used for phthalein. The value of the index of elimination places prime importance upon the ratio of excretion rather than mere quantity of excretion. Also the stability of renal function is a much truer guide of the ability of the kidneys to withstand the ordeal of major operative procedures than is the mere estimate of the quantity of dye which they can excrete at any given time. Whether or not this stability has been reached can be learned only by repeated tests, if reliance is placed upon methods depending upon quantitative determination. The index of elimination furnishes evidence of this stability from a single observation. Experience has shown that patients with an index of one or more, or who, in other words, are excreting an equal or larger percentage of dye in the first period as compared with the third, are in condition of functional stability and are suitable operative risks. Patients with an index below one or who are excreting a smaller percentage of dye in the first period as compared with the third are in condition of functional instability and operation is unsafe.

For the past five years I have used this method of determination of renal function in surgery with satisfaction for the relief of prostatic obstruction. Surgical procedures have been both suprapubic prostatectomies and transurethral resections. From my observations I have noticed that in a few instances, where the nitrogenous blood values would be well within normal figures, the index of elimination would be much below one, the interpretation being that such a patient would not have good renal stability. The dependability of this method was further accentuated by the fact that I have operated on cases with such findings, and found postoperative kidney function to be sorely tried. In some instances death resulted from renal insufficiency. In other words, a patient may have normal blood values (non-protein nitrogen, etc.) but following operation it would be found that renal stability or reserve power would be very much diminished—a fact which could easily have been determined by using Thomas' index of elimination.

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A SELF-RELEASED LOCK-BOLT FOR FRACTURES OF THE FEMORAL NECK*

BY E. W. CLEARY, M.D. AND

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A device which locks into the head fragment so firmly that the reduced fractured surfaces of the neck may be impacted securely and permanently together, is much to be desired. One of the writers (G.M.M.) has invented a device which has proved so satisfactory that this preliminary report is in order.

This instrument is a hollow bolt of stainless steel, three and one-half inches long and one-quarter inch in diameter. The penetrating end has a slot three-quarters inch long and one-eighth inch wide, holding two levers hinged on a single cross-pin. While the bolt is being inserted these locking levers lie completely within the slot. When the bolt is in place the locking levers are pushed out by a threaded pin one-eighth of an inch in diameter, which traverses the hollow in the bolt to a position transverse to the long axis of the bolt. The protruding end of this pin has a knob by which it may be easily turned. The protruding end of the bolt is fitted with a loose-fitting nut. When the lock levers are out and this nut is turned up tight against the cortex the reduced surfaces are held so firmly together that no cast, splint or other external fixation appliance is necessary. The patient may be freely turned in bed, and may be allowed up on crutches as soon as the wound has healed. When the bolt is to be withdrawn, the centre pin is unscrewed and the locking levers rotate back beyond the end of the bolt and flush with its surface, thus offering no possible obstacle to withdrawal.

Our procedure is to reduce the fracture under local anæsthesia by Leadbetter's manœuvre, check reduction by heel-palm test, and confirm by antero-posterior and lateral x-rays. These are taken with four Michel clips, so placed in the skin about the hip that the clips and their shadows in the x-rays enable us to aim the one-quarter inch drill used to make way for the lock-bolt. We expose the trochanter for the drill under local anæsthesia. X-rays check the position of the drill, which is withdrawn and the bolt immediately inserted. The bolt is locked and tightened as above described.—*Calif. & West. Med.*, 1936, vol. 45, No. 3.

Illustrations of this appliance are given in the original article.

* From the Orthopædic Service of the San Mateo Community Hospital, San Mateo, California.

Editorial

THE ALLERGIC FACTOR IN RHEUMATIC FEVER

IF we regard rheumatic fever solely as a clinical problem we would undoubtedly look upon it as a microbic infection. The discovery by Poynton and Payne[1] of the "Diplococcus rheumaticus" seemed to provide the necessary proof of the truth of this conception. But as soon as other laboratory workers attacked the subject doubt began to be cast on this simple notion. Many competent observers were unable to confirm Poynton and Payne's results in a considerable number of cases or to reproduce in animals the lesions typical of the disease. Hence has arisen one of the greatest medical controversies of our time.

It is fairly generally conceded now, however, that there is some association between streptococci in general and rheumatic fever. Unfortunately, there is no constancy in the strains of streptococci isolated. Some have recovered the non-hæmolytic *S. viridans* (Rosenow,[2] Swift and Kinsella,[3] Swift and Boots[4]); others, strains that produce no changes on blood (Poynton and Payne,[5] Small,[6] Birkhaug[7]); others, hæmolytic strepto-

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4. SWIFT, H. F. AND BOOTS, R. H.: Influence of sodium salicylate upon arthritis of rabbits inoculated with non-hæmolytic streptococci, *J. Exper. M.*, 1923, **37:** 553.

5. POYNTON, F. J. AND PAYNE, A.: Researches on Rheumatism, Churchill, London, 1933, p. 122.

6. SMALL, J. C.: Bacterium causing rheumatic fever and preliminary account of therapeutic action of its specific antiserum, *Am. J. M. Sc.*, 1927, **173:** 101.

7. BIRKHAUG, K. E.: Rheumatic fever: bacteriologic studies of non-methæmoglobin-forming streptococcus, with special reference to its soluble toxin production, *J. Infect. Dis.*, 1927, **40:** 549.

cocci (Gray, Fendrick and Gowen[8]). To make the matter still more complicated, Rosenow has been able to transform a green streptococcus into a hæmolytic or indifferent type by altering the conditions of growth, and some have not been able to find any microorganisms in the blood of many rheumatic patients (Coburn[9]). Birkhaug[10] claims to have isolated a specific streptococcal toxin to which he attributes the manifestations of acute rheumatism, but his conclusions have not been accepted.

In view of the lack of unanimity in regard to the specificity or otherwise of any of the several strains of streptococci that have been isolated from the blood of rheumatic patients and the contradictions in regard to the presence of antibodies therein the majority of investigators seem to have fallen back on the theory of allergy. Notable supporters of this view are Coburn (*loc. cit.*), Poynton and Schlesinger,[11] Collis,[12] and Swift.[13] The theory, briefly stated, is that in a non-fatal infection the responsible microorganisms are attacked by the tissue cells and broken down. During the process the body may become sensitized to the disintegration products, consisting largely of nucleoprotein, and then will react in a characteristic fashion should a fresh invasion and disintegration of the same or a related organism take place. Ample opportunity is afforded, of course, because of minor throat infections, for the tissues to become sensitized, and particularly to streptococci. In as much as streptococci are to be found in the throats of most people, at least in the more northerly climes, and often cause no obvious damage, we must take into account the soil as well as the seed. Granted a hypersensitive state of the tissues, a "trigger" may set off the explosion—a fresh infection, rapid changes in temperature, dampness, unsuitable food, poverty, chronic ill-health, nervous strain, race, or heredity. There may not be, necessarily, one exciting cause only; there may be a combination of several.

In illustration of the rôle played by infection we may cite the curious relationship that can be traced between rheumatic fever and some other diseases. Apart from the frequently symptomless character of streptococcal infection of the naso-pharynx, there are some diseases which are associated, though less obviously than rheumatic fever, with a streptococcal sore throat. Thus, rheumatism and rheumatic endocarditis can often be dated from a previous attack of scarlet fever, measles, influenza, or pneumonia. Moreover, these other affections are even more frequently the incentive for a severe relapse in subjects who are mildly or definitely rheumatic.

We may conclude, then, with considerable certainty, from the evidence available, that a close relationship exists between streptococci (using that name in its widest sense) and rheumatic fever, but we must agree also that the evidence does not entitle us to state that rheumatic fever is either a bacteriæmia or a toxæmia. The most plausible explanation, therefore, appears to be some form of allergy.

Considerable evidence may be adduced in favour of this theory. A suggestive parallel may be drawn between rheumatic fever and tuberculosis, the latter disease, as is admitted, being often accompanied by allergic manifestations. Both affections may remain latent for years and then, either gradually or suddenly, make themselves obtrusive. In the young rheumatism, like tuberculosis, commonly assumes a generalized form; later in life, when a certain degree of immunity has been established, the lesions in both cases tend to become localized, and so we have phthisis and chronic articular rheumatism, fibrosis being prominent in both. The serous membranes in both are favourite sites for trouble. In childhood the nervous system is particularly vulnerable, as witness tuberculous meningitis and chorea. The familial incidence in both diseases is very similar and the clinical course in both is not

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unlike. The Heberden's nodes of rheumatism and the erythema nodosum of tuberculosis suggest allergy. Finally, the response of rheumatic patients to the intravenous injection of the products of streptococcus growth is similar to that of tuberculous persons to tuberculin.

As Weintraud[14] and others have pointed out, the rheumatic state presents many clinical resemblances to the well-known "serum sickness"—the fever, the local œdemas, the skin eruptions and arthritic pains—in short, to allergy. Variations in response may and do occur, apparently dependent on the nature of the provocative organism or organisms and the degree of vulnerability of the tissues. Experimental evidence has been adduced by many observers, notably, Derick, Swift and Andrews,[15] Zinsser and Grinnel,[16] and Dochez and Stevens,[17] which shows that artificially induced hypersensitiveness varies in animals at different stages. At first the animal becomes allergic only to the original sensitizing organisms, but later the allergy becomes more general in quality, and reactions will occur with different strains of the same organism. The behaviour of rheumatic fever seems to conform with these observations; a condition possibly produced in the first place by a specific streptococcus might, quite conceivably, be caused to relapse or recur from the stimulus of various other strains of streptococci, or even from that of unrelated microorganisms or even non-bacterial proteins.

As is well known in the case of anaphylaxis, after the preliminary sensitization there is a silent period of ten to twenty-one days, after which a larger dose of the antigen will precipitate the anaphylactic reaction. It is worthy of note that Schlesinger[18] found, in his study of rheumatic children at a convalescent home at West Wickham, England, who had recurrences, the silent period which elapsed from the provocative tonsillitis (lasting 2 to 5 days) to the onset of the rheumatic manifestations, either arthritic or cardiac, was also from ten to twenty-one days. The same holds good for the arthritis that sometimes follows scarlet fever.

Contrariminded are Aschoff,[19] who thinks that the lesions of serum-sensitization do not accurately reproduce those of rheumatic fever, and Opie,[20] who says "Evidence that sensitization to products of the many types of streptococci is an essential precursor to the occurrence of rheumatic fever is as yet lacking." Opie, however, admits that allergic phenomena modify its course. A.G.N.

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REFORMING THE MEDICAL CURRICULUM

THE problem of reforming the medical curriculum is one that is ever before us. Faults can be found with our existing systems and all are agreed that the time is ripe for a change. Indeed, the advances in medical science in all fields are so enormous that it is quite impossible for any one mind to keep abreast of them. It is unfair and, indeed, futile, to expect students to do so. The length of time allotted to the medical course has been materially increased of late years and is generally regarded as being at its limit. The difficulties attending the planning of a proper medical curriculum are great at the moment and will become greater as time goes on. If we cannot increase the length of the course we have to face the only other alternative, *viz.*, to so teach and reorientate the subjects already laid down in such a way as to get greater value from them. There seems to be general agreement that the desiderata are that the medical student should be more cultured, that his powers of observation and reasoning

should be cultivated, and that he should evince sound commonsense. He should be fitted, speaking generally, to take a worthy and leading place in the life of his community.

At the moment the lines along which the reform of medical teaching is being directed seems to concern the pre-medical training of the prospective medical student, the better correlation of the pre-clinical subjects, and the more effective linking-up of the pre-clinical and clinical branches. This, at least, was the trend of thought at the recent meeting of the Association of American Colleges held at Atlanta, Ga.

This conference was of special interest to Canadians for several reasons. The University of Toronto, McGill University, and the University of Manitoba have been members of the Association for some years, but their number was added to by the admission of Queen's University, the University of Western Ontario, and the University of Alberta. Then the Association honoured Dr. E. Stanley Ryerson, of the University of Toronto, and Canada in his person, by electing him President for 1936-37. By a strange coincidence, when the meeting of this Association was last held in Atlanta, forty years ago, Dr. (later Sir William) Osler, another Canadian, was chosen President. And, finally, papers were presented by three Canadians, namely, Dr. J. C. Meakins, of McGill, Dr. William Boyd, of Manitoba, and Dr. George S. Eadie, Professor of Physiology at Duke University, who is a graduate in medicine of Toronto.

A brief reference to some of the more important papers will be in order. Concerning the pre-medical education of the student the titles of two papers, "Liberal Values in Pre-Medical Education", by Dr. E. E. Reinke, Professor of Biology, Vanderbilt University, and "The Liberal Arts Background for Medicine", by Dr. Ralph J. Gilmore, Professor of Biology in the University of Colorado, are indicative of the tendency to get away from the more purely technical training in this period of the students' education and to increase the cultural aspects by a greater liberalizing of the instruction given to pre-medical students. Both speakers emphasized that scientific subjects might be taught in such a manner that the student might increase his cultural at the same time as his scientific knowledge.

Members of the Departments of Surgery, Obstetrics, Medicine, Pædiatrics, and Psychiatry of Emory School of Medicine, in Atlanta, described their teaching methods and objectives. No attempt is made at this institution to route the student through each of the special branches into which Internal Medicine is being subdivided, but the student comes into contact with all these special fields by following particular cases to them when they are referred for consultation. They considered that not sufficient time is being devoted to psycho-biology, psychiatry, and the psychoses.

Four of the five papers read on the third morning of the conference constituted a symposium on "The Integration of the Medical Curriculum". In the first of these Dr. G. S. Eadie described their method of conducting the course in Physiology at Duke University. Each week a type case is taken, say, a hemiplegia, and the general features of it are demonstrated to the class. The students are then required to work up the physiological, anatomical, histological, and embryological features which are appropriate to the condition. The other departments are informed as to the type of case studied each week, and they cooperate by providing opportunities for the student to cover the case from their particular viewpoint. The students are also required to read the relevant literature. At the end of the week the class is brought together again and the whole subject is reviewed. This method has been in operation for several years and has proved very successful.

Prof. William Boyd, in his paper entitled "Self-help in the Teaching of Pathology", expressed the opinion that the object of the course in Pathology was to enable the student to gain an understanding of disease processes and their relation to clinical manifestations, and not to teach him to differentiate in detail each type of pathological change. To this end he had evolved a new type of museum in which gross specimens are mounted under watch-glasses; photomicrographs, coloured illustrations from textbooks and atlases, and pictures of the men whose names are associated with the different diseases are grouped on one museum table, the top of which stands at an angle of forty-five degrees; the history of each case the specimens from which are displayed is in a

vertical slot on the front of the table. The subject is divided up and three students are assigned to each sub-division. A list of these sub-divisions and of the topics for each group are posted at the beginning of the year, so that each student knows what field he will be expected to prepare. When the class meets the students have to select and present in the lecture-room the museum specimens required to illustrate the subject for the day. They are also expected to look up the literature, the clinical features, and any relevant anatomical or physiological points. The particular group of students then presents the subject before the class and are questioned by the Professor. In order that the whole class will come prepared with some knowledge of the subject for the day three students are called out of the class at random and are questioned as to their general familiarity with the subject. This scheme is working admirably.

Dr. J. C. Meakins, of McGill, dealt with "The Integration of Clinical Medicine with the Pre-clinical Sciences". After an admirable philosophical analysis of the importance of the essential unity of the medical course and of the coordination of its component parts he gave some information in regard to the method adopted by him for the correlation of the allied subjects of importance to his department of Medicine.

The "Confession of Faith" of the Association is conveyed in the following excerpt from the new section 3 of its by-law on Curriculum which was adopted:—

> The entire course should be designed to train the student in the practice of medicine, including the cultivation of health, and the prevention and treatment of disease.
>
> The main purpose of the under-graduate curriculum should be to provide the student with a sound foundation in the fundamentals of medicine on which he can build in the future in general or special practice or in scientific investigation. He should have acquired such habits of mind and thought that in addition to profiting by his professional experience he will continue to educate himself throughout his life. By the end of under-graduate medical courses the student should have matured sufficiently to assume the responsible duties of his profession.

A.G.N.

Editorial Comments

A Clinical Study of Silicosis

We have from time to time published researches into the highly important but obscure subject of silicosis. So far these researches have been largely carried out on the experimental and laboratory side. Now we are able to present our readers with some work on the clinical aspect. Drs. Moore and Kelly have under their observation a large number of men in the Hollinger Gold Mines at Timmins, all of whom are exposed, though in varying extent, to the hazard of inhaling silica. Workers in all mines of this type (in Canada) are always good material for study, but it is not often that the opportunity is taken advantage of so fruitfully as in the present case.

In brief, these two authors have made an extremely careful and thorough investigation into aspects of silicosis which we believe have not yet received any attention. The important point is brought out that in silicotics the basal metabolic and pulse rates are consistently lowered, whilst the sedimentation rate is raised. The immediate practical bearing of this is that it may be possible from data of this nature to recognize potential silicotics, since it was shown that the findings were equally applicable to those in the stages of fibrosis preceding the silicosis.

We hope that this pioneer work will be followed up by others. It need hardly be said how welcome is any light on the problem of such a crippling disease. H.E.M.

Nasty Noises in New York

Apropos of the anti-noise campaign which is being waged in many cities we may draw attention to what is being done in New York under the driving force of Mayor La Guardia. The movement in that city began about sixteen months ago, and was at first a movement of persuasion, the noisy members of the community being asked to please be quieter. Last April the police issued 9,288 warnings, while summonses and arrests totalled 547. In May legal enactments were adopted which provided fines for offending. The following were prohibited:—sounding automobile horns except as a danger signal; playing the radio or musical instruments loudly enough to annoy neighbours; keeping noisy pets; blowing steam

whistles, except as a signal; using engined vehicles without a muffler; bellowing by street hawkers; the use of loud-speakers by theatres, stores, auction rooms or peddlers for the purpose of attracting attention; the noisy loading or unloading of trucks; the use of clanging or clattering vehicles; and the use of amplifiers on cars or trucks except with police permission. After a time public interest seemed to wane and there was not so much cooperation. In November the police issued 15,661 warnings, and the number of arrests and summonses jumped sharply to 1,012. It is expected that the process of education will be slow, but the fine system is apparently producing results; it is noticed already that an offender rarely is apprehended a second time. As might be expected, automobiles continue to be the worst offenders against peace and quiet, and radios come next.

In addition to police activity complaints are investigated by the various Departments. For example, the Health Department looks into complaints in its own field. Each complaint is the subject of a written report by the man who investigates it, and his report goes to the City Hall. The report forms the basis of the reply to the person making the complaint. Similarly, the Sanitation Department investigates complaints about its trucks and the reckless throwing about of garbage and ash cans. The plan apparently works well and is beginning to bear fruit. A.G.N.

Special Articles

PNEUMOTHORAX WITH MASSIVE COLLAPSE FROM EXPERIMENTAL LOCAL OVER-INFLATION OF THE LUNG SUBSTANCE*

BY CHARLES C. MACKLIN, F.R.S.C.

University of Western Ontario Medical School, London, Ont.

During the fixation of living lung substance by the insufflation of osmic acid vapour in air through a catheter introduced into the right lower lobe of the lung of an anæsthetized cat, the animal died with the chest in permanent dilatation, which proved to be due to a double pneumothorax with massive collapse of the lungs. The autopsy findings, and particularly the suggestion of air-paths through the pulmonic interstitial tissue, were of sufficient interest to prompt a number of repetitions and variations of the original experiment with the object of clearing up the obscure points. It was soon apparent that the osmic acid had nothing directly to do with the pneumothorax, which was evidently related to the over-inflation of a limited part of the lung. A typical case will first be described.

Typical case.—The animal, usually a mature cat, is lightly anæsthetized with chloroform, and then given 2 c.c. of Dial "Ciba" liquid compound intraperitoneally. It shortly becomes completely relaxed. While stretched upon an operation board, an ordinary fairly stiff "woven silk" male urethral catheter is inserted into the trachea and gently pushed down as far as it will go. The No. 12 French size (about 2 mm. calibre and 4 mm. in external diameter) was most used. In many cases entry was through the glottis but latterly the tracheotomy route was preferred. A current of air was then passed into the catheterized part, which, on account of the anatomical arrangement, was always the right lower lobe or fraction thereof. The air, from the compressed air mains of the building, first passed into a large pressure bottle, and from there by flexible rubber tubing to the catheter. An interpolated branch led off from this hose to an ordinary mercury manometer of U-tube type, for pressure registration.

The effect of such an insufflation, provided that the pressure be high enough, will obviously be to over-distend the part of the lung cannulated by the catheter tip at the expense of neighbouring parts of the lung. This over-distension will stretch all the parts implicated —bronchi, bronchioles, respiratory bronchioles, alveolar ducts and alveolar sacs. In ordinary conditions each alveolar wall or base is bolstered up by the equal pressure in neighbouring chambers or by connective tissue, but here the pressure is unduly great in the territory receiving the blast of air, so that there is undue strain upon this part of the lung tissue. Evidence of damage is seen histologically in the fibrinous exudate, often containing blood cells, which gathers in the alveoli. A tight joint could not, of course, be made between the catheter and the bronchus in this way, and thus there must have been some reflux of air around the catheter tip, but the arrangement served for the desired ends.

Direct evidence that we are in this way over-distending the part was afforded by x-ray, in two special cases in cats, through the kind cooperation of Dr. Paul M. Andrus, Roentgenologist of the Queen Alexandra Sanatorium, Byron, Ontario. When 4 c.c. of uroselectan B (Schering) was first put into the lung area about to be insufflated through the catheter the fluoroscope showed clearly the dimensions of the part. When air was being blown in this part was seen to become appreciably en-

* Read at the Fourth Federated International Anatomical Convention at Milan, Italy, in September, 1936.

larged, and after the collapse its dimensions became even smaller than at first. Permanent records of these three stages—normal inflation, over-inflation, and collapse — were made on ordinary x-ray films (Fig. 1).

The picture of the animal during the experiment varies considerably, depending on the air pressure used and the duration of insufflation. If the pressure is high a swelling of the abdomen and chest is quickly seen, with immediate cessation of respiratory movements. The swelling of the chest is often abrupt when the "break-through" from the mediastinum occurs. If the pressure is low the enlargement of the abdomen and chest comes on slowly and almost imperceptibly, and external evidence of injury may be afforded only by the stopping of breathing. If insufflation be continued sufficiently vigorously the root of the neck swells, and later the axillæ, as air extends through the connective tissue out of the superior mediastinum along the course of the great vessels, and if the input be still continued the whole side of the animal swells by extension of air down from the axillæ in the superficial fascia, and the body becomes tense.

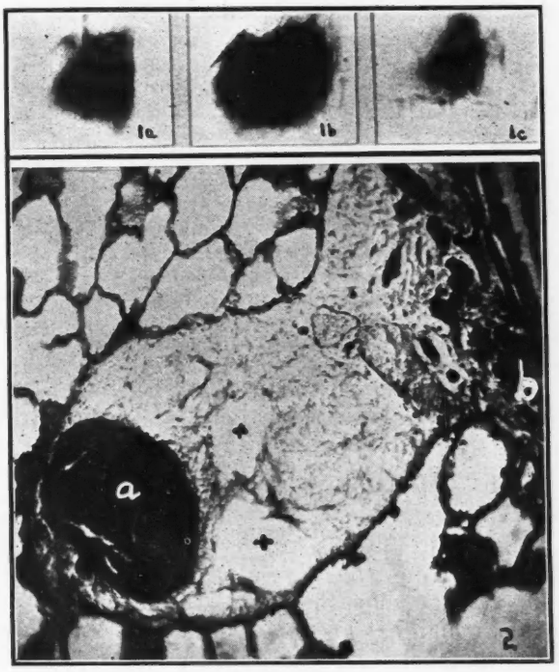

Fig. 1.—The shadow of the uroselectan in the insufflated right lower lobe of a cat, as seen in a radiograph by Dr. Paul M. Andrus of the Queen Alexandra Sanatorium, Byron, Ont. The region has been partially filled with uroselectan B (Schering). (1a) Shows normal degree of inflation; (1b) shows over-inflation, the result of insufflation of air, in this case by the use of a hand bicycle pump; and (1c) shows collapse of this lobe. The roentgenographs were taken in sequence as lettered, one almost immediately after the other, on the same animal, S-63-35. **Fig. 2.**—Swollen sheath of small branch of the pulmonary artery containing air (+). Artery (2a) contains blood; bronchiole (2b) shows no thickening of sheath. The layer of alveolar bases surrounding the sheath is seen. Magnification x 66. This picture is taken from the insufflated lobe of a cat's lung, S-63-30R-4P-6. The air, here, is comparatively small in amount.

Autopsy.—If the chest be opened immediately, air escapes, showing that there was a positive pressure within the pleural cavity, but this was not great, and is soon equalized. The walls then fall in slightly. Both lungs are totally collapsed, and most of the space of the pleural cavities is occupied by air. Another very striking feature is the large amount of air in the form of conspicuous bubbles which occupies the mediastinum, greatly enlarging this partition, although it does not bulge laterally nearly so much after death as it does before rupture. Medial to the apices the foamy structure encroaches markedly upon the pleural cavities. A peculiar and characteristic frothy appearance is given to the coverings overlying the heart because of the air bubbles which have infiltrated them. The œsophagus is enveloped in froth, and so too is the descending aorta, and bubbles may be traced down to the openings of these structures in the diaphragm. The foam has extended into the roots of the lungs of both sides, and when the vessels of these are probed their sheaths, particularly those of the arteries, are found to contain the bubbles.

On opening the tense abdomen, the retroperitoneal region contains large bubbles of air. The kidneys characteristically float upon great blebs two or more inches across. The air often invades the pelvis. If the air-flow has been prolonged a rupture of the peritoneum often occurs, and then some of the air is in the peritoneal cavity and escapes when the abdomen is opened; in such cases the retroperitoneal air is not nearly so evident. By milking down the bubbles from the mediastinum while the retroperitoneal region is submerged they may be seen to enter the abdomen along the course of the aorta and, to a lesser degree, along the œsophagus. It is quite evident that the air in the abdominal cavity has come in from the mediastinum at these points. Foam has been found filling the lesser omentum. In extreme cases foam occupies the root of the neck, extending up from the superior mediastinum along the course of the great vessels, and even out into the axilla. Cases were seen where the input of air was moderate, and where there were no bubbles of air in the neck.

Lung examination.—The lungs were examined in three states: (1) fresh, immediately after the operation; (2) after fixation in the collapsed condition; and (3) after fixation by filling the bronchial tree with fixing fluid and so distending the lungs to approximately the normal degree of inflation.

The insufflated lobe shows the perivascular sheaths, particularly those of the arteries, dilated with air. A good way to examine the part is to fix in the inflated condition, and then, after allowing the tissue to stand in fixative for a day or two until quite stiff, to cut it into thick slices transversely, beginning at the hilus. Examination of each slice is made with the binocular microscope. The pulmonic blood vessels in typical cases are collapsed and may even appear as if suspended by threads of tissue, the surviving filaments of the perivascular sheaths, to the outer layer of the sheath. The latter is thus separated from the blood vessel in the centre by a great space filled with air in the fresh condition. In the fixed tissue the air may disappear, leaving the space filled with fluid (Fig. 3). By proceeding in this way the lobe may be examined in serial slices from the hilus to the periphery. It has, from such studies, been established that these artificial air passages, the result of air-dissection during the insufflation, are continuous from the peripheral, small branches of the blood vessels to the hilus, where the air-spaces become continuous with the large bubbles in the mediastinum. The extent of this cannulation, and indeed of this interstitial emphysema in general, in the mediastinum

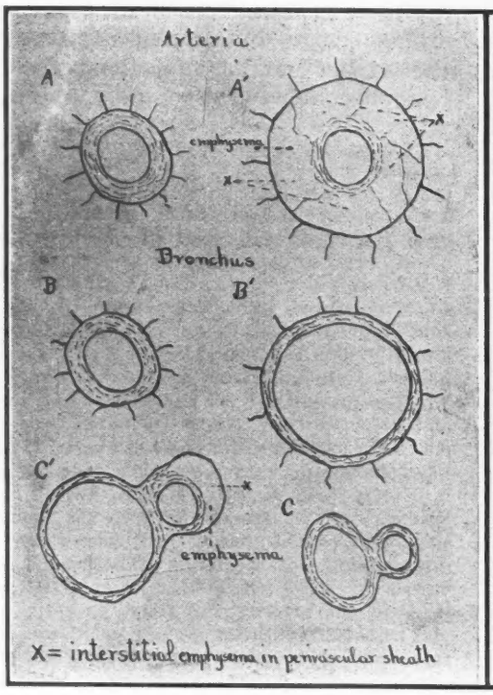

Fig. 3

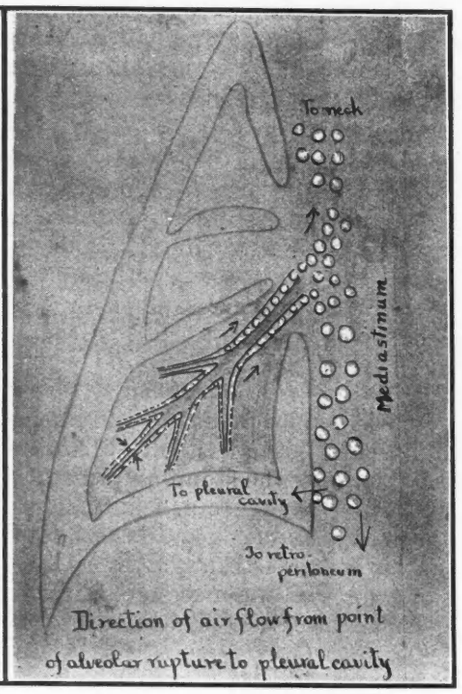
To neck
Mediastinum
To pleural cavity
To retro-peritoneum
Direction of air flow from point of alveolar rupture to pleural cavity

Fig. 4

Fig. 3.—Diagram to show swelling of sheath of pulmonary arterial branches in the insufflated lobe, in contrast to the thinning of the wall of the air tubes under the same conditions. A, B and C represent the approximate normal, and A^1, B^1 and C^1 show the insufflated condition. Note the thickening of the arterial sheath in A^1 as contrasted with the normal sheath in A, and also the invasion of air "x". The air-tube sheath in B^1 is thinner than that of the normal, and its substance supports the alveolar bases, shown between the radiating partitions, while in the case of the artery the alveolar bases are deprived of their normal support on account of the change in the underlying tissue of the sheath. See text. C represents both artery and bronchiole, and their condition in insufflation in C^1. **Fig. 4.**—A diagram to show the pathway of air from the air-spaces of the lung to the mediastinum and beyond, via the perivascular sheaths of the lung. Only the artery is represented. Bubbles of air in the sheaths, and connective tissue of the mediastinum are shown as high-lighted spherules. One of the many points of invasion of the perivascular sheaths is indicated by the arrows in the lung interior; and one of the sites of break-through into the pleural cavity is also indicated by an arrow.

and extensions as well as in the lung, depends largely on the forcefulness with which the insufflation was done. In moderate cases there may be but relatively little air in the interstitial tissue of the lung, while in extreme cases it is very evident. Fixation in the collapsed state has the effect of shrinking the tissue and diminishing the size of the artificial air-spaces, thus rendering these passageways obscure.

Blocks of tissue were cut from the different parts of the lung, embedded in paraffin and celloidin, sectioned and stained. Serial sections were prepared from paraffin blocks. Many frozen sections were made and stained.

A curious feature is the thickening of the finer perivascular sheaths. The impression is that these have become infiltrated with fluid, presumably from the capillaries, for the contents show fibrinous material and blood cells, particularly red disks. The appearance suggests that the outer boundary of the sheath, made up of the approximated bases of the adjacent pulmonic alveoli (Fig. 2), has been enlarged during the experiment, attended by diminished pressure in the sheath; and the influx of fluid has been apparently prompted by the necessity for the re-establishment of equilibrium. Air bubbles were found in such dilated sheaths, leading to the conjecture that air is breaking through here into the zone of presumed lower pressure in the interstitial tissue from the adjoining alveoli. The lymphatics are dilated, frequently, in the insufflated zone.

Point of leakage of air from the air-chambers into the interstitial tissue.—It seems likely, then, that air in finely subdivided form breaks through from the alveoli abutting upon the perivascular sheaths into this perivascular tissue, which has become swollen and succulent, and thus presumably weakened, through the imbibition of fluid. This break-through occurs during the insufflation, when the region of lung is over-distended, and when presumably as a consequence there is a pressure gradient leading from the air-chambers into the underlying connective tissue. At first this seems paradoxical, and it would appear that a plus pressure in the insufflated area would involve all parts of that area equally, and that, accordingly, the pressure in the perivascular sheaths would be raised coincidently with that in the air-chambers. But if that be so how can we account for the presence of excess fluid, and air, in these sheaths? We have visualized the blood-vessel sheaths as covered in, or bounded,

by a membrane consisting of the approximated bases of pulmonic alveoli. These bases collectively form a system of branched tunnels in which lie the blood vessels. When hyper-inflation occurs it seems evident that actual enlargement of this circle of alveolar bases occurs; indeed it appears that then the ring of expanding bases of the alveoli is acting like the inner circle of an expanding ring-shaped rubber balloon (A and A[1], Fig. 3). In such a doughnut-shaped balloon, the hole becomes larger when the balloon is blown up to larger size, and the boundary of the hole is increased. The rubber of this inner boundary is actually being stretched, and the space within enlarged. So, too, in the case of the membrane of alveolar bases bounding the perivascular spaces, during hyperinflation, it is being stretched, and this occurs by pull of the stroma, attending the over-dilation of the alveoli, and the space within — the perivascular space — is enlarged, with coincidental diminution of pressure. Fluid from the stretched capillaries of these stretched alveolar bases thereupon transudes into the lower-pressure zone of the perivascular sheath, and air also breaks through. There is reason to think that this break-through of air occurs rather suddenly throughout the insufflated portion of the lung. Under normal conditions there is, of course, no pressure gradient from the air-spaces into the perivascular sheaths, and the alveolar bases do not leak air. My proof of leakage at this point is not final; I have not yet seen any actual openings from the alveoli into the sheaths through which the air could have passed. Doubtless they are very small, and close up after the air has passed through, so that they are difficult or impossible to see in stained sections. But globules of air, or the spaces which they formerly occupied, have frequently been found in these sheaths (Fig. 2), and their presence is difficult to explain in any other way than by the assumption that they have broken in through the alveolar bases. Collectively the boundaries of the sheaths of the insufflated region would form a considerable area through which air would leak into the interstitial tissue, which would be large enough, in my opinion, to account for the large volume of air which has accumulated in these cases in the mediastinum and beyond.

It is interesting that the interstitial tissue surrounding the bronchial tree contains no air in these cases, unless there is a pulmonic artery or vein adjacent (Fig. 3, C and C[1]). This may be explained by the fact that these air tubes are all dilated under the conditions of the experiment, and their sheaths are thus stretched and thinned (Fig. 3, B and B[1]). Thus the physical conditions in the air-tube sheaths are quite different from those in the pulmonic blood vessels, and there can be no pressure gradient between the alveolar bases surrounding the air-tubes, and the underlying interstitial tissue.

Transfer of air through the interstitial tissue.—We assume, then, that the insufflated air breaks through at myriads of points in the periphery of the perivascular sheath system of the insufflated part of the lung. The condition for this must be that the local pressure be sufficiently high, for if it is relatively low the air may escape, provided only a part of a lobe is being insufflated, through the pores leading into adjoining lung territory (Macklin[4, 5, 6, 7]). But if the blast be too great to be released through the pores, or if an entire lobe be thus cannulated, then there is air-leakage into the perivascular sheaths as described. The minute globules coalesce into large ones, and these all move toward the hilus, passing along lines of least resistance, in the direction of the larger parts of the vessels, where the interstitial space is greater. Thus, with prolonged insufflation, we may visualize a continuous streaming of air bubbles from many finer into fewer coarser channels, the minute bubbles of the finer sheaths constantly coalescing so that bubbles of larger and larger size are formed. The air-stream dissects its way to the root, where it occupies the sheaths of the great pulmonic vessels and then invades the mediastinum, which it distends and traverses to invade adjoining territory as shown. It even ruptures the mediastinal wall (Fig. 4).

The mode of production of pneumothorax.—It is possible to have interstitial emphysema of the insufflated lobe without pneumothorax, where the insufflation is moderate and the volume of air which gains admittance to the interstitial tissue is relatively small so that it does not burst the mediastinum. But with continued insufflation and transfer of air into the mediastinum the latter may swell so that the delicate wall ruptures at the point of least resistance, and discharges the air into the pleural cavity. In this way we have a pneumothorax with massive collapse, which becomes complete if the insufflation be continued. In cats and rabbits rupture into one pleural cavity was followed by the collapse of both lungs, on account of the contralateral pneumothorax which supervened upon the breaking through of the thin mediastinal septum. Where the blast of air is free there is a rapid swelling of the entire thorax following the bursting of the mediastinum.

In one case the point of rupture was found on the posterior mediastinal wall of the left pleural cavity, just above the diaphragm; and here the break was made plainly evident by the stream of bubbles which emerged from it after water had been added to the opened thorax to such an extent that the point of exit was sub-

merged, air being meanwhile continuously injected from the original point of insufflation, which in this animal (a rabbit) was the superior mediastinum. A glass cannula had been introduced about three-quarters of an inch into the superior thoracic aperture, and after the insufflated air had led to double pneumothorax and massive collapse a filtered suspension of Tiemann's blue in normal saline was injected through the same medium, to mark the course of the air and its point of entry into the pleural cavity. Thus localized, the existence of the orifice was easily confirmed by observation of the stream of bubbles which emerged from it on further insufflation. In another rabbit the point of exit from the mediastinum was indicated by coloured fluid on the wall of the right pleural cavity, but confirmation here by outbubbling into water was not attempted. It may be that the position of the point of invasion of the pleural cavity may differ widely in different animals, even of the same species, and further studies are being made as to it. We may conclude that the pneumothorax is consequent upon the bursting of the overstrained mediastinal wall. The actual bulging of the wall was witnessed directly in the rabbit just mentioned where the point of exit of air was accurately localized in the opened thorax. When air was insufflated into the superior mediastinum the wall of the mediastinum, particularly that part behind and below the level of the heart root, was seen to widen very markedly each time a jet of air was injected, before it was liberated through the rupture. We are thus fairly certain of the point of entry of air into the pulmonic interstitial tissue, and quite certain of its general region of exit into the pleural cavity.

Additional experiments.—We must admit the possibility that air may be leaking directly out of the insufflated lobe through the pleura, thus causing the pneumothorax. This has been considered very carefully. In two cases where coloured butyl alcohol was run into the right lower lobe through the catheter it was found in small quantities in the right pleural cavity after the insufflation and collapse, showing that when the lung is damaged, as it must have been by this reagent, it becomes permeable to fluid and doubtless air also. In other cases where coloured fluid was in the part being subjected to the air-blast evidence of some leakage through the pleura was found, but even here there was evidence of transfer of air through the pulmonic interstitial tissue, as seen in the air-spaces in the perivascular sheaths. But in the typical cases, where no fluid was introduced into the lung, we have no evidence that air was going from the lung through the pleura directly into the pleural cavity, and good evidence that it was not. In one such case, after pneumothorax had followed insufflation as described, we removed the lung and inflated it to at least the degree of normal inflation while submerged in water, and no streams of air bubbles were to be seen; hence we concluded that no artificial breaks had been produced in the pleura. A second experiment was performed similarly, also in a cat, and the lungs were inflated as fully as possible. No rupture of the pleura in the insufflated lobe was to be detected; not a bubble of air issued from the submerged lobe. Indeed, when finally the entire lung was inflated to the bursting point only two air-streams were to be seen, and both of these were in the non-insufflated part of the lung—one in the upper right lobe and the other in the middle left. Even if air were passing directly out of the lung into the pleural cavity it is difficult to understand how such a striking degree of mediastinal emphysema could follow, and be produced by the pneumothorax. In taking such a view we would have to explain why the mediastinum bulged into the pleural cavities when the pressure in them, on this assumption, would have to be greater than that in the mediastinum. But to assist us in settling this point we performed an artificial pneumothorax on a rabbit by injecting air under pressure of 2 cm. of mercury through one of the lower intercostal spaces directly into the pleural cavity, and though we obtained a double pneumothorax by this manœuvre there was no emphysema of the mediastinum. Another similar experiment on a cat produced bilateral pneumothorax, but absolutely no mediastinal emphysema. In this instance a pressure of 12 cm. of mercury was used. Air in the retroperitoneum, axilla and inguinal region was found, attributable to direct out-break from the pleural cavity, but none in the mediastinum. Hence it is felt that the air is leaking into the pulmonic interstitial tissue and through that it gains entry into the mediastinum; that it gets into the pleural cavity from the mediastinum, rather than that it invades the mediastinum from the pleural cavity.

In a case (rabbit) where air was directly injected into the superior mediastinum, and where meanwhile the diaphragm was being directly observed, the abdomen having been opened, it was noted that bulging of the area of the diaphragm bounding the inferior end of the mediastinum occurred first, and that after this the lateral parts of the diaphragm bulged. This sequence of events was interpreted to mean that the pleural cavities were filled with air from the ruptured mediastinum, and the point of rupture of its wall was afterwards located exactly, as has already been described. Thus, while we could not force air from the pleural cavity into the mediastinum we could force it from the mediastinum into the pleural

cavity. Mediastinal emphysema consequent upon emphysema of the pulmonic stroma thus assumes great importance, for it may lead directly to pneumothorax, and also to emphysema of the abdomen, neck and axilla.

An effort was made to trace the point of inlet of air into the interstitial tissue and the exit thereof from the mediastinum into the pleural cavity by the use of coloured fluids introduced into the insufflated part of the lung. It was hoped that the fluid would be carried with the air into the interstitial tissue, and would not only mark the point of inlet but also the general course through the interstitial tissue as well as that through and out of the mediastinum. It was found, however, with all fluids tried that they were held with remarkable tenacity in the lung substance, and it could not be demonstrated that they had even entered the interstitial tissue, much less points beyond. This was true of diluted India ink, suspensions of Tiemann's blue in water and normal saline, and methylene blue in a solution of sodium taurocholate, and other fluids. The x-ray studies showed that there was no spread, beyond the local area into which it was injected, before or following massive collapse of the lung, of the uroselectan B used to define clearly the area under increased local pressure.

General Remarks

An interesting feature of the experiments was the infiltration of air from the mediastinum into the interstitial tissue of the uninsufflated portion of the lungs. In some cases air was found in the perivascular sheaths of the uncatheterized lobes of the right side, and also even in the lobes of the left side. It seems probable that this extension of the mediastinal emphysema occurred before the collapse, for after this has occurred the pressure gradient between mediastinum and lung would not be so steep as it was before.

As to the pressures used, these ranged from one centimetre of mercury or less to a rare extreme of twenty-two centimetres. In general the time required to produce a rupture of the mediastinum and consequent pneumothorax in this way varied inversely as the pressure used.

Catheters of smaller size than No. 12 were used occasionally, but when that was done it was more difficult to produce pneumothorax. This was accounted for, in part at least, by the assumption that when the stream of air was introduced into a subdivision of a lobe there was opportunity for the air to escape peripherally through the pores into adjoining territory of the lung, and so prevent such marked over-distension as when the blast involved almost, if not quite all of, the right lower lobe (Macklin[4, 5, 6, 7]).

An effort was made to find out whether or not the breaking-point for air from the air-chambers might be facilitated by gases, such as osmic acid vapour in the insufflated air, or by fluids of low surface tension, such as solutions of the bile salts. It is too early to make definite statements on this subject but it does seem as if conditions of surface tension play a part in the retention of air. This problem of the mechanism of confinement of air within the air-spaces of the lungs, and, conversely, of the protection of the interstitial tissue from invasion by air, is of fundamental importance.

Clinical Applications

It is not proposed here to enter into a lengthy discussion of possible correlations of these experiments with the vast record of clinical experience in massive collapse and pneumothorax. It may be that in the human subject conditions develop which permit of air-invasion of the pulmonic interstitial tissue in such quantities as to lead to mediastinal dilatation, and if this occurs it may be that rupture would supervene as in these experimental cases, with attendant collapse of the lung, partial or complete. We are, of course, dealing in our experiments usually with extreme cases of pneumothorax and collapse, and have used intrapulmonic pressures which are greater than those possible in clinical experience. This mode of production of pneumothorax may account for the failure in so many cases to find evidence of a rupture on the surface of the lung. It must be confessed that one would hardly be likely to look for a rupture on the mediastinal wall; and these ruptures, in my experience, are very difficult to locate after the bleb which gave rise to them has collapsed, and where no coloured fluid, or air current, is used to determine the exact site. It will suffice now merely to ask if massive collapse with spontaneous pneumothorax may not arise clinically in the following manner. In a lung in which a main bronchus has become plugged with mucus, air may still be admitted to the plugged lobe in inspiration because of the widening during that phase (Macklin[3]) of the air-tube, which allows air to slip past the plug but prevents it from returning because in the expiratory phase the tube narrows. Escape of the trapped air to other parts of the lung through the pores may be impossible (Macklin[4, 5, 6, 7]), either because it is a lobar bronchus which is blocked or because the pores are occluded. Pressure may then be assumed to rise in the blocked region, due to accumulation of air which cannot be absorbed as fast as it is admitted. The alveolar bases might then be thought to become pervious to air and allow it to escape into the perivascular sheaths, as in the experimental cases.

Transfer through the interstitial tissue to the mediastinum, accumulation therein, and rupture into the pleural cavity would ensue. Such a pneumothorax and massive collapse would be unattended with any marks of rupture through the pleura; and we find records of such failures to find outlets from the lungs in the literature (LeWald[2]). Such a mechanism would not involve a cough, and indeed we find from the clinical histories that some of these cases of spontaneous pneumothorax accompanying massive collapse come on in the patient's sleep, when no violent respiratory effort is being made. We do find them in patients who give a history of some type of pulmonary involvement, however, so that there is scope for such a mechanism to operate as that hypothesized above. This explanation would not apply, of course, to those cases where there is an obvious rupture of the lung's surface, but only to those cases where there is pulmonary interstitial emphysema and no external rupture. It is in just such cases that LeWald feels that there must be a third type of massive collapse, caused neither by atelectasis nor by rupture of the pleura.

Some of these clinical cases of spontaneous pneumothorax give evidence of acute abdominal distress, so marked in some cases as to indicate the need of an abdominal operation. Reduction of the pressure in the thorax affords relief to the abdominal condition. These patients may have abdominal pain due to the downward pressure of the dome of the diaphragm caused by high intrapleural pressure; and they may have an actual pneumo-peritoneum and pneumo-retro-peritoneum, as we have found in the experimental animals. Evacuation of the air from the pleural cavity allows the dome of the diaphragm to rise, and air from the retroperitoneal tissues to flow back into the mediastinum with its reduced pressure, and thence out into the pleural cavity, to be taken out by the aspirating needle. The dyspnœa which the patients experience with the onset of a large pneumothorax may be due not only to the lessened air flow but to the impeded action of the heart caused by the compression of its circulation by the large air blebs in the layers between the pericardium and the pleura. Embarrassment to the mediastinal circulation through pressure of the air-accumulation was conspicuous in our experimental animals, leading to circulatory arrest.

Cases have been reported where pneumoperitoneum has been induced during the operation for artificial pneumothorax production. We did not find retroperitoneal air when the pressure was as low as 2 cm. of mercury—a higher pressure than one would use clinically. Ten cm. hæmoglobin did, however, produce retroperitoneal air, in our experience. Is it possible, in the clinical cases, that the needle punctured the lung—a possibility by no means remote (Howlett *et al.*[1])—in a region held by adhesions to the pleural wall, in a case where free outlet of air through the bronchi was not possible? In such a case, insufflation into the lung substance would occur, and the conditions of our experimental animals be duplicated, to some extent at least. The pressure in these cases need not be so high as in the experimental animals with tissue unweakened by disease. Is it too much to imagine that in such cases pulmonary interstitial emphysema would be produced artificially and that the train of consequences, pneumo-mediastinum with rupture of the walls causing pneumothorax and massive collapse, and also extension of air into the peritoneum and even into the neck, would supervene?

I wish to thank those who have collaborated so kindly, in so many ways, in gathering together these results; Mr. Walter Downs, for assistance with the experiments, sections, and photographs; Dr. Paul M. Andrus and his assistant, Mr. S. A. Olheiser, for the roentgenological observations and records; Dr. David W. Crombie, Superintendent of the Queen Alexandra Sanatorium, and the Directors of that Institution, for opportunity to utilize the x-ray appliances of the Mara Research Laboratories; and last, but not least, Dr. Madge Thurlow Macklin, for her constant and invaluable aid.

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HYPERPROLANURIA AND PATHOLOGICAL PREGNANCY.—K. Heim states that although the Aschheim reaction has proved its worth as a diagnostic test for pregnancy in the past ten years the hopes that it might be used quantitatively in the problems of pathological pregnancy have not altogether been fulfilled. In cases of hydatidiform mole and chorion epithelioma the values of prolan A and B in the urine may be increased or decreased. In hyperemesis gravidarum and other toxæmias of pregnancy hyperprolanuria is the rule. When chorion epithelioma occurs apart from pregnancy the quantitative test is of value in estimating recovery and the formation of secondary growths. Heim believes that there is an etiological connection between hyperprolanæmia and retroplacental hæmorrhage, for several cases have been published of hæmorrhage in early pregnancy with hyperprolanuria in which at biopsy no hydatidiform mole has been present. He has observed the same thing in 12 cases of ante-partum hæmorrhage with hyperprolanuria. In 25 cases of eclampsia hyperprolanuria was present. The amount of prolan excreted must be estimated in relation to the amount of urine excreted, whether this be normal or diminished and highly concentrated. In the latter case prolan may be found in citrated blood and cerebro-spinal fluid. The source of prolan production is the placenta, and a definite ratio between the quantity of prolan excreted and the placental mass has been proved in multiple births.—*Med. Welt*, Dec. 26, 1936, p. 1868. Abs. in *Brit. M. J.*

Medical Economics

Medical Relief in the Province of Ontario

Medical relief throughout the Province of Ontario has now been under the jurisdiction of the Ontario Medical Association for a period of two years, the present plan having commenced as from March 1st, 1935. Prior to that date there were many conferences between the Minister of Public Welfare and the Ontario Medical Association at which the entire situation relating to the problem of supplying medical care to relief recipients was considered. As there was no precedent of the operation of a similar plan elsewhere it was necessary to devise a system which would provide not only for the financial administration of medical relief funds but also for some method of handling the monthly accounts from approximately 3,000 doctors who would provide medical care to some 400,000 relief recipients. After carefully considering the various alternative methods of administration, it was finally decided that complete responsibility be vested with the Ontario Medical Association, towards which end an Order-in-Council was passed setting out the provisions whereby the medical relief funds would be provided and payment made to doctors for their services and the supplying of medication to recipients of relief. There being no guiding precedent, as noted above, it was arbitrarily agreed to conduct the experiment on a monetary basis of 25 cents per month for each person registered on relief in all municipalities in Southern Ontario, the per capita rate provided in the Territorial Districts being 50 cents per month. It was further agreed that certain commonly used medical supplies classified as "Household Remedies" be obtained through the regular channels of Relief Administrators, the cost of which supplies would be borne by the municipalities over and above the amount provided for medical care. In order to facilitate matters it was agreed that the monthly payment of medical relief moneys to the Ontario Medical Association would be made through the Provincial Welfare Department, the amounts chargeable against each municipality being deducted from the monthly amount of assistance refunded to each municipality on the part of the Department of Public Welfare.

The next step towards setting up the administrative machinery was the appointment of Medical Relief Committees throughout the Province. These Committees were formed for the purpose of scrutinizing and taxing the monthly accounts rendered by physicians in respect of each municipality, and the Committees were comprised of doctors appointed by the County Medical Societies. The local Relief Officer and a druggist representative were also included upon each Committee. Whereas there were over 600 individual municipalities in southern Ontario it was provided that one Committee might preside over more than a single municipality, which resulted in the appointment of some ninety Medical Relief Committees. In northern Ontario, the doctors elected to form two pools only, each conforming with the geographical Ontario Medical Association Districts in that section of the Province. Complete authority was vested with each of the Medical Relief Committees in the matter of taxing the monthly accounts, even to the extent of disciplining those doctors in whose cases it was apparent that abuses were in evidence. All matters of policy, however, and particularly those of a financial administrative nature, were left to the Central Relief Division of the Ontario Medical Association.

Having completed arrangements for the set-up of the "field" machinery, attention was then given to the establishment of the Central Administrative Office. Towards this end, after consultation with our auditors, it was decided to accept their proposal whereby they would loan one of their Chartered Accountants to organize this particular phase of the administration. Within the limited time at our disposal the Head Office was organized, a staff engaged, office equipment purchased, and the various forms were printed for use by the doctors and Medical Relief Committees in rendering their monthly medical relief accounts for payment. In setting up the records provision was also made for the compiling of certain statistical information, a comprehensive summary of which is contained in the published Annual Report of the Medical Relief Division as of April 30, 1936, being the conclusion of our first year's operations.

In review, it may be pointed out here that the average percentage of 38.1 paid to the doctors in Old Ontario, resulting from the monthly per capita rate of 25 cents per person on relief, also included the entire cost of drugs supplied by the doctor to relief recipients. As from March 1, 1936, arrangements were made with the Ontario Retail Druggists' Association whereby the cost of drugs supplied would be covered by a deduction from the 25 cents per month paid by the Government, amounting to 4 cents, or 16 per cent, which latter amount was turned over to the Ontario Retail Druggists' Association for distribution to the participating druggists. These arrangements, however, were

applicable to the cities and some of the larger towns only throughout the Province, about 100 places being included. As a result of the two years' operation of the existing medical relief plan, during which period well over $2,000,000 of medical relief funds have been distributed, considerable experience has been gained, and there are a number of refinements which it is proposed to introduce, which in our judgment will strengthen our already smoothly working plan.

To conclude, the administration of medical relief, as vested with the Ontario Medical Association, may be briefly summarized as follows.

1. The number of complaints made to the Government on the part of the relief recipients have been negligible, which clearly demonstrates to the Government that the Ontario Medical Association is capable of administering medical relief in an efficient and satisfactory manner. This administration has been carried on exclusively by the Ontario Medical Association without any supervision whatever on the part of the Government.

2. The administration of medical relief by the Ontario Medical Association, on behalf of the doctors of the Province, has proved to be entirely satisfactory to the medical profession, apart from the amount paid to the doctors for their medical relief services, as noted below.

3. It has been definitely established that the amount provided, *viz.*, $3.00 per year per person on relief, is entirely inadequate to cover the cost of the limited medical services rendered, not overlooking the fact that no provision has been made to take care of surgery and hospital services.

N.B.—A new contract has just been completed between the Government and the Ontario Medical Association for a two-year period March 1, 1937, to March 1, 1939, at a new per capita rate of 35 cents. T.C.R.

Association Notes

THE ANNUAL MEETING

The Canadian Medical Association

President, H. M. ROBERTSON, Victoria;
President-elect, T. H. LEGGETT, Ottawa;
General Secretary, T. C. ROUTLEY, Toronto.

The Ontario Medical Association

President, W. K. COLBECK, Welland;
President-elect, R. K. PATERSON, Ottawa.

Ottawa Medico-Chirurgical Society

President, H. B. MOFFATT, Ottawa;
President-elect, R. L. GARDNER, Ottawa;
Secretary, T. L. FISHER, Ottawa.

Since the last number of the *Journal* went to press considerable progress has been made in the organization work at Ottawa.

As an indication of the growing enthusiasm among the members of the local society, its annual banquet, held at the Royal Ottawa Golf Club on the evening of February 18th, was the most largely attended in its history. On this occasion the guests of honour were Dr. T. H. Leggett, and Dr. R. K. Paterson, Presidents-elect, respectively, of the Canadian and of the Ontario Medical Associations. Also present as guests were Dr. T. C. Routley and his assistant, Dr. Kelly, of Toronto. It is interesting to note that since the dinner membership in both major organizations, so far as Ottawa and district are concerned, has grown by leaps and bounds.

From time to time the various sub-committees of the Committee on Arrangements are submitting reports which amply justify the assurance already given that the combined sessions to be held in Ottawa next June are bound to uphold the best traditions of the medical profession in Canada. In consideration of the very large attendance which is expected, the high standard of the scientific program, and the arrangements made for a hospitable welcome, one is justified in believing that the week's proceedings will mark a great achievement for the three associations.

In former "Association Notes", the activities of the Scientific Exhibits Committee under the Chairmanship of Dr. George S. Williamson were alluded to. It should be mentioned, once again, that all those desiring to offer exhibits in the section devoted to this purpose should communicate with Dr. Williamson, 295 MacLaren Street, Ottawa, without delay. Already sixteen exhibits have been fully arranged for as follows:—

1. Title—Pulmonary Tuberculosis, the Importance of X-Rays in the diagnosis of, and Collapse Therapy in the Treatment of. Institution—Toronto Hospital for Consumptives, Weston, Ontario.
   Exhibitor—L. W. Thompson, M.D.
2. Title—The Landmarks of Late Heredosyphilis. Institution—Hôtel-Dieu Hospital, Montreal.
   Exhibitor—Paul Poirier, M.D.
3. Title—Physiological Standardization of Drugs. Institution—Department of Pensions and National Health, Laboratory of Hygiene, Ottawa.
4. Title—Technique of Block Anæsthesia. Institution—Mayo Foundation, Rochester, Minn.
   Exhibitors—J. S. Lundy, M.D., L. H. Mousel, M.D., E. B. Tuohy, M.D. and R. C. Adams.
5. Title—Neuropathology. Institution—Division of Neuropathology, University of Toronto, Toronto.
   Exhibitor—Eric A. Linell, M.D.
6. Title—Roentgenographic and Photographic Exhibit from the Montreal Neurological Institute of McGill University.
   Exhibitor—J. N. Petersen, M.D.

7. Title—Parasitic Worms of Man in Canada. Institution—The Institute of Parasitology, McGill University, Montreal.
   Exhibitor—Thomas W. M. Cameron, M.D.
8. Title—The Causes of Normal and Abnormal Hæmorrhage from the Uterus. Institution—225 Avenue Road, Toronto.
   Exhibitor—Melville C. Watson, M.D.
9. Title—Cerebral Pneumography in Childhood. Institution—Montreal Neurological Institute, McGill University, and Children's Memorial Hospital, Montreal.
   Exhibitor—A. E. Childe, M.D.
10. Title—Deficiency States Due to Disease in the Gastro-Intestinal Tract. Institution—Mayo Foundation, Rochester, Minn.
    Exhibitors—Albert M. Snell, M.D., and Dwight L. Wilbur, M.D.
11. Title—A Study of the Human Spine. Institution—Queen's University, Kingston, Ont.
    Exhibitors—W. A. Janes, M.D., and R. C. Burr, M.D.
12. Title—Series of Aneurysms of the Aorta and Heart. Institution—Pathological Department, Queen's University, Kingston, Ont.
    Exhibitor—James Miller, M.D.
13. Title—Surgery of the Chest. Institution—University of Toronto.
    Exhibitor—Robert M. Janes, M.D.
14. Title—An Exhibit on Radiology. Institution—Toronto General Hospital, Toronto.
    Exhibitor—G. E. Richards, M.D.
15. Title—Pneumonia Treatments and Care. Institution—Metropolitan Life Insurance Company, Canadian Head Office, Ottawa.
16. Title—(To appear later). Institution—Department of Health and Public Welfare, Winnipeg.
    Exhibitor—F. W. Jackson, M.D.

Comparatively few of the spaces reserved for commercial exhibits remain. Fifty spaces have already been taken up, and applications are steadily coming in for more.

On Wednesday, June 23rd, His Excellency the Governor-General will be the guest of honour at luncheon. He has kindly consented to be present at the ceremonial function also, which will be held on the evening of the same day and which will be attended by ecclesiastical, military, and political dignitaries, civic officials, and other persons prominent in many walks of life in Canada.

On Thursday, June 24th, it is expected that the Prime Minister, the Right Hon. W. L. Mackenzie King will be the guest of honour. A speaker for the Friday luncheon has not yet been selected. The luncheons to be held daily throughout the course of the meetings will be of the service club type, the tickets selling at one dollar.

Arrangements for the garden party to be held on the afternoon of Thursday, the 24th, are well under way.

The Committee on Golf report that on the afternoon of Friday, June 25th, the tournament for the Hamilton and Ontario trophies will be played. Provision has been made for the teeing off on this occasion of no fewer than 600 players. Ample opportunity will be given for taking care of any overflow at Ottawa clubs other than those whose facilities have already been requisitioned. After the tournament a special entertainment will be provided at the Chateau Laurier for all golfers and their friends, and at this function the prizes will be presented.

"The Junior Committee" reports that all visitors comprising the Junior Group, namely, those younger persons who will visit Ottawa with association members, will be properly entertained during their sojourn in the capital. On Wednesday afternoon, June 23rd, there will be a city sight-seeing tour. On Thursday afternoon a feature of the entertainment will be a boat trip on the Ottawa river. The program for Friday morning will include a swim in the Chateau pool. together with an exhibition of diving, and for the afternoon, visits to the Royal Mint, the Victoria Memorial Museum, the Parliament Buildings, and the Dominion Archives.

Dr. J. H. Alford, 235 O'Connor Street, Chairman of the Committee on housing, continues to urge the necessity for all prospective visitors making arrangements for hotel reservations forthwith.

The following is a list of the invited speakers who have to date signified their intention of presenting papers at the forthcoming annual meeting at Ottawa.

SECTION OF ANÆSTHESIA

Dr. Harold Griffith, Montreal.
"Anæsthesia from the patient's point of view."

Dr. John S. Lundy, Rochester.
"The present unusual opportunities for medical men in anæsthesia."

Dr. C. H. Robson, Toronto.
"Anæsthesia in children."

Dr. H. J. Shields, Toronto.
(Subject to be announced).

SECTION OF DERMATOLOGY

Dr. F. J. Burgess, Montreal.
(Subject to be announced).

Dr. D. E. H. Cleveland, Vancouver.
(Subject to be announced).

Dr. H. A. Dixon, Toronto.
"The past year at the skin clinic, Hospital for Sick Children, Toronto."

Dr. L. P. Ereaux, Montreal.
"Facts, fads and fancies in the treatment of acne vulgaris."

Dr. W. R. Jaffrey, Hamilton.
"Disturbances of the sebaceous glands." (lantern slides).

Dr. Harold Orr, Edmonton.
"Urticaria."

Dr. Antonio Sabetta, Ottawa.
"Tuberculosis verrucosa cutis and cancer alike."

Dr. B. Usher, and
Dr. A. D. Campbell, Montreal.
"Perineal and pudendal nerve resection in the treatment of pruritus vulvæ."

Dr. Geo. S. Williamson, Ottawa.
"Kaposi's varicelliform eruption. Report of a case with experimental evidence in support of a virus etiology."

Dr. Norman Wrong, Toronto.
"The treatment of recurrent erythema multoforme."

Dr. E. J. Trow, Toronto.
(Subject to be announced).

SECTION OF MEDICINE

Dr. D. M. Baltzan, Saskatoon.
"Serious vascular accidents not commonly diagnosed."

British Speaker (Name to be announced later).

Dr. Maurice Brodie, New York.
"Immunological problems of poliomyelitis."

Dr. Geo. Hale, London.
"Nephritic œdema; its causes and treatment."

Dr. K. A. MacKenzie, Halifax.
"Hyperpiesia."

Dr. W. E. Ogden, Toronto.
"The abolition of clinical tuberculosis by anticipation and control; a definite scheme and its application."

Dr. Geo. Young, Toronto.
"Early diagnosis of carcinoma of the stomach."

SECTION OF OBSTETRICS AND GYNÆCOLOGY

Dr. H. B. Atlee, Halifax.
"Pre-natal and post-partum treatment."

Dr. H. C. Burgess, Montreal.
"Abortion."

Dr. Ernest Couture, Ottawa.
"Sterility."

Dr. Geo. Hooper, Ottawa.
"Endometriosis."

Dr. John Mann, Toronto.
"Obstetrical complications."

Dr. J. D. McQueen, Winnipeg.
"Menorrhagia of the menopause."

Dr. Wm. Scott, Toronto.
"Treatment of genital prolapse."

Dr. W. P. Tew, London.
"Pre-eclamptic toxæmia."

Dr. Melville C. Watson, Toronto.
"The endocrine factors in normal and abnormal menstruation." (lantern slides).

SECTION OF OTO-LARYNGOLOGY

Dr. G. E. Hodge, Montreal.
"Stricture of the œsophagus."

Dr. A. H. Veitch, Toronto.
"Discomfort in the throat: diagnosis."

Dr. D. E. S. Wishart, Toronto.
"Petrous tip suppuration."

Dr. J. B. Collip, Montreal.
Dr. Hector Mortimer, Montreal.
Dr. R. P. Wright, Montreal.
"Symposium—Naso-genito relationship and the treatment of atrophic rhinitis with œstrogenic substances."

SECTION OF OPHTHALMOLOGY

Dr. Wm. Lowrey, Toronto.
"Some experiences with detachment of retina."

Dr. C. E. O'Connor, Kingston.
(Subject to be announced).

Dr. F. T. Tooke, Montreal.
"Cardiovascular hypertension as revealed in the fundus oculi."

SECTION OF PÆDIATRICS

Dr. Alan Brown, Toronto, and
Dr. D. E. Robertson, Toronto.
"Differential diagnosis and treatment of the acute abdomen."

Dr. A. E. Childe, Montreal.
"Roentgenology, especially in pædiatrics."

Dr. H. B. Cushing, Montreal.
"Rubella or German measles."

Dr. Donald Fraser, Toronto.
"Immunization in childhood."

Dr. Alton Goldbloom, Montreal.
"Icterus neonatorum."

Dr. A. M. Jeffrey, Ottawa.
"Childhood tuberculosis."

Dr. H. M. Keith, Montreal.
"The treatment of epilepsy in children."

Dr. Perry Long, Baltimore, Md.
"The clinical use of para-amino-benzine sulphonamide or its derivatives in the treatment of beta-hæmolytic streptococcal infection."

Dr. Howard Spohn, Vancouver.
"The physiology of the central nervous system in regard to pædiatrics."

Dr. F. F. Tisdall, Toronto.
"The present status of vitamin therapy in childhood."

SECTION OF RHEUMATIC DISEASES

Dr. W. S. Barnhart, Ottawa.
"Canadian organization to combat rheumatic diseases."

Dr. F. S. Burke, Ottawa.
"Report of a Canadian survey of rheumatic diseases."

Dr. A. A. Fletcher, Toronto.
"Some problems of rheumatic diseases, national and international."

Dr. G. Douglas Taylor, Montreal.
"Differential diagnosis and treatment of chronic arthritis."

SECTION OF SURGERY

Dr. G. W. Armstrong, Ottawa.
"Certain aspects of the treatment of fractures of the forearm."

British Surgeon (Name to be announced later).

Dr. J. H. Couch, Toronto.
"Infections of the hand."

Dr. J. T. Danis, Toronto.
"The treatment of pruritus ani."

Dr. Arthur Elvidge, Montreal.
"Fractures of the skull."

Dr. Fulton Gillespie, Edmonton.
"The clinical problem of the cystic breast."

Dr. R. M. Janes, Toronto.
"Surgical treatment of pulmonary tuberculosis."

Dr. Neil J. Maclean, Winnipeg.
"Post-operative complications following abdominal operations."

Dr. Fulton Risdon, Toronto.
"Automobile injuries to the head."

Dr. Philip D. Woodbridge, Boston.
"Important minor points in local anæsthesia."

SECTION OF UROLOGY

Dr. E. D. Busby, London.
"The so-called silent renal stone."

Dr. Gordon S. Foulds, Toronto.
"A review of our bladder tumour cases."

Dr. R. C. Hastings, Quebec.
"Conservative treatment in urogenital tuberculosis."

Dr. J. C. McClelland, Toronto, and
Dr. K. F. Davis, Toronto.
"Tuberculous epididymitis: renal tuberculosis."

Dr. R. A. McComb, Toronto.
"Hæmaturia in general practice."

Dr. David W. MacKenzie, Montreal.
"Lesions of the bladder neck and trigone in the female."

Dr. Frank S. Patch, Montreal.
(Subject to be announced).

Dr. Robin Pearse, Toronto.
"Renal pain—diagnosis and treatment."

Dr. R. E. Powell, Montreal.
"Suprapubic prostatectomy, methods and results: a comparison with transurethral resections."

The following letters are self-explanatory and will be read with pleasure and satisfaction.

OTTAWA, 11TH FEBRUARY, 1937.

Sir,

I am desired by His Excellency the Governor-General to forward to you the enclosed letter from the Keeper of the Privy Purse, informing you that His Majesty King George VI has been graciously pleased to grant his patronage to the Canadian Medical Association.

I have the honour to be,
Sir,
Your obedient servant,
(Sgd.) F. L. C. PEREIRA
Assistant Secretary to the Governor-General

The Chairman,
Canadian Medical Association,
184 College Street,
Toronto 2, Ontario.

PRIVY PURSE OFFICE,
BUCKINGHAM PALACE, S. W.
1st February, 1937

Dear Sir,

I am commanded by The King to inform you that His Majesty has been graciously pleased to grant his Patronage to the Canadian Medical Association.

Yours truly,
(Sgd.) ULICK ALEXANDER
Keeper of the Privy Purse.

The Chairman,
Canadian Medical Association,
184, College Street,
TORONTO 2.

Hospital Service Department Notes

Radiologists and Hospitals Agree on Principles of Relationship

For some three years the professional status of the radiologist and the basis of his financial arrangement with his hospital have been receiving considerable discussion in radiological and hospital circles. Particularly have the bases of remuneration and the right of the hospital to make any profit from the department been questioned. Certain suggestions offered, such as the privilege and space rental basis, have been found unadaptable to many smaller hospitals or to those with excessive

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All communications intended for the Department of Hospital Service of the Canadian Medical Association should be addressed to Dr. Harvey Agnew, 184 College Street, Toronto.

non-pay patronage. A satisfactory agreement has now been effected. Some months ago a special committee of the American Hospital Association drafted proposed "Principles of Relationship" and, at a meeting in Chicago, on February 14, 1937, with representatives of the various radiological organizations these principles as finally revised were unanimously adopted by the radiologists. The Radiological Inter-Society Committee, representing the American Roentgen Ray Society, the American Radiological Society and the Radiological Society of North America, spoke for the radiologists, and was supported by the Chancellors of the American College of Radiology who were also present.

The principles agreed upon (and which immediately follow) are of considerable interest to Canadian radiologists and hospitals, represented as many of them are in the organizations participating in this conference. The essential feature of the agreement is that there must be no exploitation by either side. Moreover, no one basis of remuneration is being urged, that basis to be adopted which would best meet local conditions. The full text is as follows:

PRINCIPLES OF RELATIONSHIP BETWEEN RADIOLOGISTS AND HOSPITALS

In view of the current discussions concerning the relationship of radiologists to hospitals, and because of the desirability of protecting the public, of maintaining radiological services of high efficiency, and of safeguarding the hospitals, the hospital radiologist and the interests of the non-hospital radiologist, the following basic principles are hereby approved by the Board of Trustees of the American Hospital Association.

1. The radiological service of the hospital shall be maintained primarily for the benefit of the sick.

2. Every hospital radiological department should be under the direction of a competent radiologist, preferably a diplomate of the American Board of Radiology, or one who is working towards that objective.* If, because of size or isolation, such arrangement be not feasible some member of the general medical staff trained in radiology should be in charge and a consultation service arranged with a nearby radiologist.

3. The radiologist is entitled to recognition as a professional member of the medical staff and as head of a hospital department.

4. The preservation of the unity of the hospital and its component departments and activities is an essential administrative principle. This principle can be maintained without any infringement on professional rights or professional dignity.

5. Inasmuch as no one basis of financial arrangement between a hospital and its radiologist would seem to be applicable or suitable in all instances, that basis should be followed which would best meet the local situation. This may be on the basis of salary, commission or privilege rental, but in no instance should either the hospital or the radiologist exploit the other or the patient.

6. When an arrangement is effected whereby the radiologist of the hospital pays a rental for space and service, cares for non-pay patients, and in return retains all private fees collected, such contract should clearly cover the matter of depreciation of equipment, replacements and additions, should protect the radiologist against excessive non-pay work, and should take into consideration the "good-will" by virtue of which a large proportion of the paying clientele is attracted.

7. The American Hospital Association views with disapproval the proposal that the actual cost of films and associated overhead be separated from the professional charges of the radiologist or that the responsibility for this department be divorced from the hospital. While in many instances this would be a financial relief to the hospitals, it would probably result in frequent omission of the radiological consultation with a specialist in radiology, would mean less efficient radiological service with potential legal complications, and would tend to create difficulties with national and other organizations requiring supervision of the radiological work by a competent radiologist.

* In Canada this would read "American Board of Radiology, or an equivalent body."

Medical Societies

Academy of Medicine, Toronto

The regular stated meeting of the Academy of Medicine, Toronto, for January was held on the evening of January 5th, and took the form of the annual library and historical night. There was an excellent attendance of the Fellows of the Academy with their wives and guests. An interesting program had been prepared, and presentations were made of two portraits, one, that of the late Dr. Newton Albert Powell, a past-president of the Academy, the other, that of the late Dr. Abraham Groves, of Fergus, from the brush of Sir Wyly Grier. Dr. Powell's portrait was unveiled by his granddaughter, Dr. Rosamond McCulloch, on behalf of her father and mother, Dr. E. A. and Mrs. McCulloch. Dr. W. A. Burr, whose early life had been spent in Fergus, gave a short biographical sketch of Dr. Groves, and the portrait, the gift of Mrs. Groves, was unveiled by the Rev. R. A. Hiltz, D.D., a son-in-law.

Dr. Norman Gwyn presented, for Mr. Thomas Rolph, the gold watch which had belonged to his father, the late Dr. John Rolph. Dr. Gwyn reviewed a few of the interesting historical facts in connection with the watch which had been given to Dr. Rolph in 1834.

The Fellows were delighted to have Dr. Wilbur Harris present to receive the certificate of Honorary Fellowship recently conferred upon him by the Academy. Dr. Harris replied briefly expressing his appreciation of this high honour.

Dr. George Shanks, Chairman of the Historical Committee, gave a short talk on articles presented to the museum during the past year. Among these was one of particular interest, the gift of Dr. Shanks himself. This was a facsimile of a page from the autopsy book of the Montreal General Hospital, 1902-1903, on which Dr. John McCrae had written the following lines:

Here begynneth ye Booke of ye Deade,
wherein is fayrely set foorth ye last state
of four hundred and seventeene persones,
tht have departed this lyfe; wherein be
tabled diverse straunge and fearsome condicions
yt have ledde to ye same final ende: God
have them of his grace.

"Our lyfe is but a winter's day.
Some only breakfast, and away.
Others to dinner stay, and are fulle fedde.
The oldest man but suppes, and goes to bedde.
Large is his dette, yt lingers out the day.
He that goes soonest, hath the least to pay!!"

Dr. M. H. V. Cameron, Chairman of the Library Committee, spoke on some of the more important accessions to the Library during the year.

Dr. H. B. Anderson presented thirty books on dentistry, among these being a rare 1728 edition of Pierre Fauchard's "Le Chirurgien Dentiste", in two volumes, and W. D. Miller's epoch-making publication, "The Micro-organisms of the Human Mouth", 1890, which he had obtained for the Academy with considerable difficulty. The thanks of the Academy were expressed to Dr. Anderson for his generosity in this regard.

The first address was given by Dr. H. M. Tovell on the "Centenary of Richard Maurice Bucke, of London, Ont., 1837-1902", in which special mention was made of his work on cosmic consciousness.

Dr. T. G. H. Drake spoke briefly on his exhibit in the Journal Room of "Prints and documents relating to wet nurses of the 18th century". This splendid exhibit created much interest.

Dr. A. E. Sutton delivered a short address on "Militia surgeons of 1837", making special mention of Christopher Widmer and Peter Diehl.

On the adjournment of the regular meeting in Osler Hall, the Fellows and their guests were taken to the Journal Room where exhibits of historical interest were to be seen and where refreshments were served. GORDON S. FOULDS

The Association of Officers of the Medical Service of Canada

At the annual meeting of the Association of Officers of the Medical Service of Canada, held in Saint John in January, the following officers were elected: *Honorary President,* Hon. Murray McLaren, Lieutenant-Governor of New Brunswick; *Hon. First Vice-president,* Brig.-Gen. F. Page; *Hon. Second Vice-president,* Lt.-Col. A. H. Landon; *President,* Capt. Geo. Lyons, Moncton; *Vice-president,* Lt.-Col. V. D. Davidson, Saint John; *Secretary-treasurer,* Capt. W. O. McDonald, Saint John.

A committee was appointed to present a biography of the late Lt.-Col. G. G. Corbet. The chairman of this committee is Lt.-Col. S. S. Skinner. Following the business meeting, a lantern slide talk was presented by Lt.-Col. D. C. Malcolm. His subject was "The pilgrimage to Vimy".

The Saint John Medical Society

At the regular monthly meeting of the Saint John Medical Society, Dr. A. E. Macaulay, senior surgeon of the Saint John General Hospital, was the special speaker. Dr. Macaulay summarized some personal experiences in the treatment of cancer in the gastro-intestinal tract, with special emphasis on stomach cancer, and on cancer of the rectum. At the completion of his talk, he exhibited a motion picture film, prepared by the Lahey Clinic, demonstrating a procedure followed in the operation for resection of the rectum. A. STANLEY KIRKLAND

Abstracts from Current Literature

Medicine

Asthma. Report of the Research Clinic at Guy's Hospital for the year ended October 31, 1936. Appendix A. Published by the Secretary, Asthma Research Council, King's College, London, W.C.2.

The importance of controls in assaying any new method of treatment is stressed, as it may well be, from this clinic, where it was found that over 50 per cent of asthmatics improved under treatment consisting of the injection of normal saline. A statement included in the report is worth quoting, "The correct attitude for the patient and the therapeutic physician is one of optimism, but for the research worker is one of profound scepticism rooted in hopeful pessimism". In keeping with their research status treatment is only assessed in those patients who have passed through a complete course of non-specific treatment including remedial advice concerning diet, occupation and habits as well as drugs for relief where required. After this they are sent for a two months' course of breathing exercises. At the end of this period 58 per cent were improved, 27 per cent unimproved, and 5 per cent were worse. Specific treatment, vaccine and protein desensitization, have so far been disappointing. In the hay-fever cases treatment with inhalant proteins is more successful, even when skin reactions are not completely obliterated. As regards Monilia, infection with these fungi in asthmatics in London is negligible, and no specific cutaneous sensitivity existed.

The studies on the psychological make-up of asthmatic children showed that they were of above average intelligence, but exhibited over-anxiety, fear of failure and a dominating

tendency. There is also a tendency in these children to be affected by environmental factors.

ARNOLD BRANCH

Hereditary Pseudo-hæmophilia. Fowler, W. M., *Am. J. M. Sc.*, 1937, **193**: 191.

Numerous records have been published which describe a disease simulating hæmophilia but which appears with equal frequency in female and male and is transmitted equally by both. It is characterized by a normal coagulation time, prolonged bleeding time, both of which occur in purpura, rather than in hæmophilia, but by a normal or increased platelet count and by no undue bruising, both of which are found in hæmophilia rather than purpura. Thus the disease partakes of part of the hæmatological picture of both of these diseases characterized by hæmorrhage, but is like neither. The patient in the first family reported by Fowler had had a history for forty years of excessive bleeding, first from the rectum when 3 years of age, then from the gums from 6 to 12 when the deciduous teeth were being lost; he almost died from hæmorrhage at 11 from a minor scalp wound; began to bleed from the nose at 13, and had a continuous history of hæmorrhages from then on. At 24 he was given horse serum, which apparently checked the bleeding, but two years later another such injection was of no avail. Many transfusions of blood did not stop the bleeding. In a period of about 12 years he was in the hospital thirteen times for prolonged hæmorrhage.

He gave a history of mild bleeding in his father's two brothers, although the father was himself normal. One sister had died at 17 from uterine hæmorrhage, and a younger brother had bled to death from hæmorrhages in the gastro-intestinal tract.

Another patient with the same condition is described, and in this family 12 persons had had a history of excessive hæmorrhage. Nine were males and 3 were females. One man who married twice transmitted his tendency to bleed to children of both wives. The name thrombasthenia has been given to this condition, but the accuracy of the term has been questioned inasmuch as these patients gave no indication of lowered number of or physiologically inadequate platelets.

MADGE THURLOW MACKLIN

Surgery

Intra-abdominal Adhesions. Bogart, L. M., *Arch. Surg.*, 1937, **34**: 128.

Various substances have heretofore been used to prevent post-operative intra-abdominal adhesions, with comparatively little success. Symptoms pointing clearly to obstructive adhesions and interference with the normal physiology of the alimentary tract are indications for the operative prevention of adhesions. The pain may be more or less constant at the site of the previous operation, or it may be distant from it. Acute exacerbation of the pain, characterized by cramp-like seizures, and followed by periods of comparative freedom from pain, are quite characteristic of obstructive lesions of the alimentary tract. Vomiting may be a late symptom. One or two stools may be effected with enemas and yet obstruction may be present. It is not wise to wait for complete obstruction of the bowel before operation is decided on, nor should one wait for the symptoms of deep shock to appear before operating. Bogart's technique is to prepare the patient two or three days prior to operation with a bland non-residue diet; saline cathartics are indicated and two enemas should be given daily. To complete deflation of the alimentary tract, gastric decompression, by instituting constant gastric drainage, may have to be done. Two doses of pitressin the day before the operation, and one dose on the operating table, help to keep the bowels contracted to make the operative procedure easier. Sharp dissection of all adhesions and removal of all tags should be practised. One should refrain from using retractors intraperitoneally or from manipulating the bowel any more than is needed. Good retraction is imperative. To provide this the author uses spinal-anæsthesia. When all dissection and hæmostasis have been attended to the peritoneum is picked up with a row of forceps, and stretched upwards, and the edges brought close to each other. The author uses a prepared solution of 0.08 per cent sodium citrate in distilled water, in which 25 mg. of papain are dissolved. Of this solution, 200 to 300 c.c. are introduced into the abdomen until 1,500 c.c. are used. In 26 of the 30 clinical cases in which this method was adopted the results ranged from good to excellent.

G. E. LEARMONTH

Traumatic Rupture of the Thoracic Duct with Bilateral Chylothorax and Chylous Ascites. Brown, A. S., *Arch. Surg.*, 1937, **34**: 121.

The author states that this is the first case of bilateral chylothorax with associated chylous ascites of traumatic origin recorded in the literature. The patient, a woman, was seen by him in consultation four months following an automobile collision in which she sustained a transverse fracture of the middle third of the left humerus. A little more than two months after the accident she developed fluid in the abdomen. A few days later the abdomen was opened and a large quantity of non-odorous gray chyle-like fluid was expressed. The wound healed by first intention. Seventeen days later dyspnœa developed and fluid was found at the bases of both lungs. The chest was repeatedly

tapped on both sides. The fluid was determined to be chyle. The abdomen again became markedly distended and two litres of chyle were removed. Repeated thoracentesis was required. Brown devised and performed a new operation to provide the most direct means of access to the site of rupture of the thoracic duct without entering either the thoracic or the peritoneal cavity. In the sitting posture paravertebræ nerve block, including the tenth, eleventh and twelfth intercostal nerves, was carried out. A vertical incision was made parallel with and 3 cm. to the right of the spinous processes, from the tenth rib to the lower border of the first lumbar vertebra. Deep dissection was made through this opening until the bone of the first lumbar vertebra was encountered. Separation was carried on between the crus of the diaphragm and the body of the vertebra. A soft mass of tissue was encountered lying over the anterolateral aspect of the vertebra. This was apparently the direct site of rupture of the duct. The cystic mass communicated directly with the thoracic cavity. The patient left the operating table, but died the following day of adrenal apoplexy as demonstrated by autopsy.

G. E. LEARMONTH

Giant Cell Tumours of the Jaws. Major, S. G., *Am. J. Surg.*, 1936, **104**: 1068.

Giant-cell tumours of the jaws are rare. The etiology is unknown. Two suggestions are offered: (1) the basis is an inflammatory reaction, and (2) the lesion is a true tumour. Trauma would seem to play some part. Giant-cell tumour is a lesion of early adult life. There is no sex predilection. Symptoms are few. Pain is not a prominent feature. Pathological fracture may be the first sign. Deformity can be detected early, since growth expands the cortical bone. The expanded shell may crepitate on pressure. The mucous membrane overlying the tumour is dark red or purple. A smooth rounded mass passing evenly into normal bone is typical.

The radiological picture is characteristic. A roughly spherical or ovoid shadow with well demarcated edges is noted. The shadow presents small lobulations. Incomplete peripheral trabeculations are to be seen.

Periosteal reaction is absent. The tumour is expansile, not infiltrative. Typically, cystic and solid areas are present. The solid portions are friable, granular, and vary from yellow to dark red in colour. Extension into a joint cavity is very rare. The neoplasm may be scraped out easily with a curette.

Microscopically, the tumour is composed of many giant cells in a stroma of smaller, rounded, spindle, and polygonal cells. Various-sized blood spaces may be seen. Areas of hæmorrhage are conspicuous. Foam cells may be present.

Malignancy probably never develops in a typical giant-cell tumour, nor are true metastases seen.

The lesion must be differentiated from a dentigerous cyst, a radicular cyst, an adamantinoma, and a sarcoma. A dentigerous cyst is always associated with an unerupted tooth. The cyst is lined with stratified squamous epithelium. The finding of vital teeth in the region of the tumours precludes the diagnosis of radicular cyst. This tumour is commonest in the upper lateral incisor region and is lined with stratified squamous epithelium. Adamantinomata may be difficult to differentiate clinically. Trabeculæ usually are seen throughout the tumour, and the microscopic picture is characteristic. Sarcomata grow more rapidly, periosteal reaction is evident, and, microscopically, show the evidences of bone formation in various stages. Occasionally giant-cell tumour may be confused with carcinoma, either primary or secondary. The author suggests pre-operative radiation as a means of differentiation. If the former is present marked diminution in size will occur.

The choice of treatment lies between curettage and roentgen irradiation, or a combination. The author prefers cauterization of the cavity by high frequency current to the use of chemicals. The author advises biopsy and the use of post-operative radiation in all cases.

STUART GORDON

Obstetrics and Gynæcology

Therapeutic Abortion by Means of X-ray. Mayer, M. D., Harris, W. and Wimpfheimer, S., *Am. J. Obst. & Gyn.*, 1936, **32**: 945.

Ten years' experience with therapeutic abortions by x-ray in Mt. Sinai Hospital, New York, is reported. The operation was performed on 200 ward patients, with a clinical success in 96 per cent and an ideal success in 90 per cent of cases. The method is recommended in pregnancies of not more than fourteen weeks' duration, in women who are suffering from a serious lesion which would make surgical interruption very dangerous and who should never again become pregnant. This is particularly valid for women over thirty-five years of age. The method has no mortality and a remarkably low morbidity, but it does require the closest cooperation between clinician, radiotherapist and gynæcologist, as well as scrupulous observation, control and follow-up of the patient. If the treatment should fail, in no circumstances should the pregnancy be permitted to continue. Irrespective of amenorrhœa, patients should receive contraceptive instruction after the abortion.

The clinical picture following radiation is that of a missed abortion, with a latent interval averaging about four and one-half weeks.

ROSS MITCHELL

Pyelitis in Toxæmias of Pregnancy. Peters, J. P., Lavietes, P. H. and Zimmerman, H. M., *Am. J. Obst. & Gyn.*, 1936, **32**: 911.

Of 320 patients with toxæmias of pregnancy 41 were found to have pyelitis. No less than 11 out of 25 patients who came to autopsy with vascular or renal disease first manifesting itself in pregnancy were found to have pyelitis and hydronephrosis or their sequelæ. Out of 93 patients with pyelitis complicating pregnancy, 25 developed hypertension or œdema, or both, before the termination of pregnancy. Reasons are given for believing that pyelitis in these patients was a major etiological factor in the production of toxæmia.

ROSS MITCHELL

A Biological Test for the Diagnosis of Intra-Uterine Fetal Death. Rezek, G. H., *Am. J. Obst. & Gyn.*, 1936, **32**: 976.

Using the Schneider modification of the Aschheim-Zondek test, the author was able to obtain the dead fetus reaction with an accuracy of 95.3 per cent in a series of 86 cases. The test is positive for dead fetus reaction as long as living chorionic tissue is in contact with the maternal blood. Its value in ectopic pregnancy lies in establishing the fact of fetal death, and, since further growth is then arrested, affording information that the danger of rupture or abortion is decreased. The reaction is not positive when post-partum or diluted ante-partum urine is used for the test. While not infallible, the test is extremely valuable, and should be used in conjunction with the clinical reactions in determining treatment in a given case.

ROSS MITCHELL

Pædiatrics

Diabetes Mellitus in Childhood. Payne, W. W., *Brit. M. J.*, 1936, **2**: 960.

Treatment in diabetic children has two aims —first, to enable the child to remain happy, active and healthy, second, to prevent progression of the disease; the first is easy, the second hard. Diet should be just sufficient to allow of normal activity and growth; anything in excess is deleterious. The estimated average requirement for a diabetic child of two is 1,120 calories (equal to Holt's average normal); the figure rises to an estimated average requirement of 1,750 calories at the age of 12 (much below Holt's average normal of 2,800). A carbohydrate-fat ratio of 2–2.5 seems best, insulin requirement being usually definitely less than with equi-caloric higher fat diets. Such a diet is essentially normal, except that sugar is restricted and the actual amounts of food taken is known. Insulin is usually necessary, and must always be used in sufficient amounts to control all symptoms, even where control has been excellent. However, the author has failed to note any permanent improvement in a single case. In some cases the blood sugar is most unstable, and even three insulin doses daily do not control well; here, protamine insulin is proving of great help. Since there is marked tendency to hypoglycæmia just before the noon meal, strenuous exercise, and especially cycling, is forbidden at this time. Otherwise, every attempt is made to show the children that with their diet and insulin they are in every respect normal.

Insulin reactions in children present most varied symptoms, abdominal pain and vomiting, and sometimes sudden coma being notable. Some patients are subject to periodic bouts of vomiting, which if improperly treated will quickly lead to severe ketosis and coma. Febrile illnesses, shocks and accidents in children may all quickly have serious deleterious effects and demand special care. In all such emergencies plenty of fluids and sugar by mouth, with enough insulin to keep the urine nearly sugar-free, is all that is necessary.

It may be noted in passing that of the 71 childhood cases observed by the author in the past 10 years, 10 have died. A family history of diabetes was obtainable in 16, (23 per cent).

W. FORD CONNELL

Orthopædics

Complete Dislocation of the Knee-joint: A Report of 7 Cases with End-results. Conwell, H. E. and Alldredge, R. H., *Surg., Gyn. & Obst.*, 1937, **64**: 94.

The treatment of dislocation of the knee-joint after reduction resolves itself into treatment of the injured ligaments which support the knee-joint. The knee-joint is supported anteriorly by the quadriceps femoris muscles and tendon with their medial and lateral patellar retinacular ligaments and the patellar ligament; posteriorly, by the oblique and arcuate popliteal ligaments and the tendons of the gracilis, semitendinosus, semimembranosus, biceps femoris, and the two heads of the gastrocnemius and popliteus muscles. The medial and lateral ligaments of the knee-joint, assisted by the anterior and posterior cruciate ligaments aid in preserving the stability given by the femoral and tibial condylar articulations with their medial and lateral semilunar cartilages.

Dislocations of the knee-joint are classified according to the relation of the tibia to the femur. In order of frequency these five dislocations are found; (1) anterior, (2) posterior, (3) lateral, (4) medial, and (5) rotary. Most of them are caused by direct violence. Ligamentous injury is always present, fracture of the upper end of the tibia was present in 2 cases. Blood vessel and nerve lesions are in-

frequent and usually temporary in duration. Closed reduction by traction and counterpressure with a plaster hip spica for 8 weeks, with prolonged physiotherapy, particularly directed to the quadriceps, should be tried for a year before open reduction is attempted, except in cases of dislocated semilunar cartilages, and/or, tear in the capsule, when open reduction is usually necessary. In these 7 cases they had perfect functional results except for slight loss of flexion.

FRANK DORRANCE

An Arthroplastic Operation for Congenital Dislocation of the Hip: A Two-stage Procedure. Colonna, P. C., *Surg., Gyn. & Obst.*, 1936, **63**: 777.

A report is presented of 6 cases in children under 10 years of age in whom it appeared that closed reduction could not reasonably be expected to give even a fair functional result. Colonna obtained 6 excellent functional results.

The primary stage consisted of thorough stretching of the thigh muscles under anæsthesia, subcutaneous tenotomy of the adductor muscles, the application of a long plaster spica to the normal limb, with skin or skeletal traction of 10 to 15 lbs., gradually increased for 2 to 3 weeks, to the affected leg.

The second stage is performed when roentgenograms show the femoral head approximating the normal acetabulum. The rectus femoris tendon might be tenotomized, the fascia lata divided transversely, the greater trochanter with the gluteal tendons chiseled through and reflected upwards, the capsule defined, cut and sutured, the normal acetabulum capaciously deepened, and the head apposed. The greater trochanter is replaced and a plaster spica applied, usually with some skin traction for a week or two, in about 20 degrees' abduction, for 4 weeks. Active motion is then attempted.

FRANK DORRANCE

Radiology and Physical Therapy

Clinical Results and Histological Changes following the Radiation Treatment of Cancer of the Corpus Uteri. Arneson, A. N., *Am. J. Roentgenol.*, 1936, **36**: 461.

Because not all patients with cancer of the corpus uteri who have operable lesions can be treated by hysterectomy, and because hysterectomy alone is inadequate treatment for patients with poorly differentiated lesions, some authors have advocated radium and roentgen rays for all patients. The clinical grouping employed by Heyman is recommended as a practical grouping in evaluating the results of surgery and irradiation. The results of hysterectomy alone are slightly better than the results of irradiation alone. The greatest percentage of cures results when the two methods are used together. Pre-operative irradiation is of greater value than treatment administered after the uterus has been removed. All the tumour must receive at least 5 to 10 threshold erythema doses; with intra-uterine radium the active length of the application should extend from the external os to the top of the fundus. A course of roentgen treatment should precede the intra-uterine application of the radius, to reduce the danger of forcing viable cancer cells through the lumina of the tubes. If hysterectomy is to be performed irradiation should be given in every instance before operation. Roentgen treatment should be limited to single exposures to each pelvic field. This should be followed by intra-uterine radium.

A. STANLEY KIRKLAND

Advanced Cancer of the Head and Neck. Martin, C. L., *Am. J. Roentgenol.*, 1936, **36**: 954.

In an effort to perfect a technique of x-ray radiation suitable for use in tumour clinics in hospitals of moderate size, where standard equipment is available, the author recommends that in large areas of carcinoma about the head and neck the treatment factors given here are suitable. Using a filter of 0.75 mm. of copper and 1.0 mm. of aluminum, the dosage should be 4,200 r for areas 7 cm. in diameter, 3,600 r for areas 10 cm. in diameter, 3,000 r for areas 15 cm. in diameter, and 2,800 r for areas 20 cm. in diameter. Treatments are given daily, and it is found wise to extend the total treatment time to three weeks with the larger lesions, utilizing smaller daily doses. At times, in suitable cases, a series of radium needles may be planted in the tumour masses or sewn into the mouth or lip before the divided doses of roentgen rays are administered. A warning is given that large total doses of roentgen rays in divided doses should be restricted to cases that have not been previously treated.

A. STANLEY KIRKLAND

Anæsthesia

Nupercain Spinal Anæsthesia by the Howard Jones Method. Trempe, F., *Current Research. in Anæs. & Anal.*, 1937, **16**: 26.

The hypobaric solution of nupercain (1:1,500) has been employed in 1,500 spinal anæsthesia administrations at the Hôpital du Saint Sacrement, Quebec City, during the last five years according to the method first described by the late Howard Jones, of London. The solution is provided ready for use in 20 c.c. ampoules and is injected without withdrawal of spinal fluid or barbotage. Nupercain is injected with the patient lying on his side and the usual level is the second lumbar space. He is then turned over on to his face with the head lower than the

rest of the body and allowed to remain in this position for five minutes to thoroughly soak the posterior sensory roots in the anæsthetic solution. Finally he is turned over on his back, the head still being kept low, and the light anæsthetic solution floats upward and attacks the anterior motor roots. After the abdomen has been prepared for operation the patient is ready for the incision.

The author considers that a systolic pressure of below 110 mm. or above 175 mm. is a contra-indication for spinal anæsthesia. He combats subsequent drop in blood pressure by the injection of ephedrin, gr. ½, about ten minutes before operation. He recognizes two types of fall in blood pressure: (1) early hypotensive crisis due to the diminution of the vascular tonus through paralysis of the sympathetic vasoconstrictor, and (2) delayed hypotensive crisis or "fluid hypotension", due either to an extensive removal of cerebrospinal fluid or to a loss of fluid through the puncture.

The importance of slow injection of the rather large volume of anæsthetic solution is stressed. Otherwise a sudden stretching of the dural sac occurs which tends to result in headache. He also recommends warming the solution to body temperature before injection. The use of large quantities (up to 20 c.c.) which is frequently advised in the literature is condemned by the author. The claim is made that by the use of higher levels for puncture, *i.e.*, between the first and second lumbar vertebræ, 15 c.c. is sufficient for any operation on the stomach, biliary passages or kidney. [*Abstractor's note.*—Theoretically, a puncture at this level is safe, the cord normally ending opposite the body of the first lumbar vertebra, but there is a case on record where definite injury to the cord, causing permanent paralysis, resulted from a puncture at this level and consequently the space between the second and third lumbar vertebræ must be regarded as the highest permissible puncture level. See "Recent Advances in Anæsthesia and Analgesia", by C. Langton Hewer, J. & A. Churchill, London, 1932, page 113.]

The importance of keeping the head and trunk lower than the rest of the body at all times is pointed out, and the danger is illustrated by a case requiring stimulation with carbon dioxide for 4 to 5 hours following the raising of his head and shoulder for a few seconds. The patient should be returned to his room by means of a wheel stretcher inclined in slight Trendelenburg position, and the foot of the bed should be kept elevated for seven or eight hours and then a strictly horizontal position maintained for 24 hours.

The author claims that the anæsthesia has been uniformly successful with the exception of a very few cases where it was incomplete, probably due to faulty puncture. Complications are rare, a transient abduceus (oculo-motor) paralysis having been noticed a few times. The fatalities which have occurred cannot in justice be attributed to the spinal anæsthetic as the sole factor.

F. ARTHUR H. WILKINSON

Therapeutics

Adequate Dosage in the Specific Serum Treatment of Pneumococcus Type I Pneumonia. Finland, M., *Am. J. M. Sc.*, 1936, **192**: 849.

The author's observations are based on a study of 219 cases of Type I pneumonia treated with serum in the wards of the Boston City Hospital between November, 1929, and May, 1935. Of the 219 patients given Type I serum 40 died (a mortality of 18 per cent). This is contrasted with 289 controls not treated with serum, 113 of whom died (a mortality of 39 per cent). Of the cases with positive blood cultures 44 per cent of the non-serum patients died, as compared with a mortality rate of 37 per cent in the serum-treated cases.

The author believes that the commonest causes of failure of serum in type I pneumonia are: (1) errors in typing; (2) insufficient dosage, or doses not given sufficiently close together; (3) the beginning of focal pneumococcic complications; (4) mixed infections of the lungs; (5) other complicating infections, and (6) the extent of the pulmonary lesion. The following rules are laid down for the treatment of uncomplicated cases of Type I pneumonia. (1) When treatment is begun on or before the 5th day of the disease, and the blood culture taken before the first injection of serum is sterile, and the pulmonary lesion is limited to one lobe, a dose of 75,000 units is adequate, provided this entire amount is given within a few hours. (2) Under the same conditions but with more extensive pulmonary involvement 150,000 units should be given. (3) When treatment is begun on or before the fifth day and the blood culture is positive a dose of 150,000 units is adequate if only one lobe is involved, or 200,000 to 250,000 units if the lesion involves more than one lobe before treatment is begun. These amounts of serum may prove ineffective if their administration is spread over two or more days. (4) When treatment is begun after the fifth day only a small percentage of cases are likely to derive sufficient benefit from the antibody to warrant its use. In these cases the dose of serum should be much larger than in comparable cases treated earlier in the disease. (5) Empyema may be prevented by early and adequate specific treatment.

E. S. MILLS

Observations on the Effectiveness of Protamine Insulin. Richardson, R. and Bowie, M. A., *Am. J. M. Sc.*, 1936, **192**: 764.

The authors point out that the more important observations which have been made about

protamine insulin are as follows. (1) It acts more slowly than regular insulin, so that if given shortly before a meal the blood sugar will be found to rise considerably after the meal prior to the insulin becoming effective. (2) It acts for a much longer time, so that as long as 24 hours after the injection the blood sugar may be normal or even below normal, thus preventing an early morning rise of the blood sugar. (3) It is accompanied by fewer hypoglycæmic reactions because of the slowness of its action, and hypoglycæmic symptoms when present are slow in development, affording a longer period before severe symptoms make their appearance. (4) It steadies the blood sugar, tending to prevent wide fluctuations from hour to hour. (5) It shows its full effect only after three to five days from the time of the first injection. Richardson and Bowie believe that it is possible to reduce both the number of units and of daily injections in diabetic patients on a balanced diet and at the same time to maintain the blood sugar at a lower and more constant level. They believe that patients who have been controlled with less than 40 units of regular insulin may be transferred to protamine insulin without hospitalization. As regards diet, one-fifth of the carbohydrate allowance should be at breakfast and two-fifths at each subsequent meal, thus allowing the metabolic burden to coincide with the delayed action of the protamine. Such patients are usually placed on two doses of protamine insulin daily, five-sixths of the daily dose being administered in the morning and one-sixth 12 hours later.

E. S. MILLS

Inhalation Therapy. Lockett, M. F., *Brit. M. J.*, 1936, **2**: 1251.

Strong claims have been made for the administration of drugs by inhalation. The tracheo-bronchial mucous membrane is said to have great absorptive power, and this seems to be supported experimentally. Fleiner, for example, found that India ink was extremely rapidly absorbed from the trachea, carbon particles reaching the lymph nodes in 12 seconds. Heubner showed that a dyestuff was taken up from the trachea of a cat enough to stain the muscles and many of the viscera within a maximum of 15 minutes. Atomized liquids have also been tested, and both aqueous and oil solutions have been found to be quickly absorbed by the tracheo-bronchial membrane.

Tests of these claims in human subjects show no impressive evidence of such absorption. No proof could be obtained of any absorption of potassium iodide in normal and diseased persons, even after long periods of inhalation. Insulin showed similar lack of absorption. Adrenalin showed more evidence of absorption, by its effect on blood pressure. On the other hand, inhalation was apparently of some value in administering certain medicaments in cases of chronic pulmonary congestion. The details given are rather too vague to determine whether there was any marked advantage by this method, but apparently there were satisfactory results in many cases.

This method, apart from the uncertainty of effect noted, presents two main difficulties,—(1) the giving of a therapeutic dose of a drug in an inhalation of moderate length, and (2) the exact measurement of the dose given.

H. E. MACDERMOT

Pathology and Experimental Medicine

Clinical Studies on the Influence of Certain Drugs in Relation to Biliary Pain and to the Variations in Intrabiliary Pressure. Butsch, W. L., McGowan, J. M. and Walters, W., *Surg., Gyn. & Obst.*, 1936, **63**: 451.

The authors have previously demonstrated that pain following cholecystectomy is due to an increase of pressure in the common bile duct. Patients who had a T-tube inserted into the common bile duct for drainage consented to act as subjects for observation. A U-shaped manometer with float and writing point which contacted a smoked drum gave readings of the intrabiliary pressure, or height to which the float rose, and of the perfusion pressure, or height to which the float rose before the resistance of the sphincter of Oddi was overcome allowing escape of the fluid into the duodenum. X-rays of the common bile duct filled with brominol gave further evidence of the mode of action of the drugs used.

After injection of morphine, gr. 1/6, the intrabiliary and perfusion pressures rose in 2½ to 5 minutes from zero to 20 mm. of water to 200 to 300 and to 400 to 450, respectively. This increase in pressure persisted more than 2 hours. The same effect, but less rapid and less marked in extent, occurred with pantopon, gr. 1/3, dilaudid gr. 1/32, and codeine gr. 1. The effect is produced by contraction of the sphincter of Oddi. Damming back took place into the hepatic ducts. Amyl nitrate, by inhalation, produced an immediate decrease of the pressures to normal, with prompt relief of pain, epigastric distress, and subscapular pain, but the pressure soon returned to its previous height. Glyceryl trinitrate, gr. 1/100, sublingually, and theophylline ethylenediamine, grs. 4, intravenously, produced a less marked but more prolonged decrease of pressures, with gradual return to the previous height. Practically all other known antispasmodics were used without any effect whatsoever. Morphine and other opium alkaloids are not indicated in pain following cholecystectomy.

FRANK DORRANCE

Variations of Blood Pressure in Diseases of the Hypophysis. Maranon, G. and Domenech, F., *Brit. M. J.*, 1936, **2**: 851.

The authors have carefully analyzed 138 well-selected and confirmed pituitary cases with a view to determining whether there is any constant relationship between various pituitary disorders and alterations in the blood pressure. Cases of basophil adenoma of the pituitary were not included, since it is the opinion of the authors and of some others that the high blood pressure usually seen in this syndrome is not due to any hypophyseal change but to the concurrent hyperplastic lesion of the suprarenal glands. After an analysis of their data, one must agree with their conclusion that, according to the evidence presented, changes in the internal secretion of the hypophysis have no influence whatever on the blood pressure. States of both hyper- and hypo-function of both anterior and posterior pituitary were included in the series and showed no consistent or uniform changes of blood pressure. The variations observed were evidently the normal differences observed in any other group of patients; the higher pressures, in each of the diseases studied, were found amongst the older patients, while pressures for the average age coincided exactly with average figures for that age.

W. FORD CONNELL

Radial Artery Changes in Comparison with those of the Coronary and other Arteries. Sappington, S. W. and Cook, H. S., *Am. J. M. Sc.*, 1936, **192**: 822.

The authors have studied the atheromatous changes occurring in various arteries in a series of 86 patients ranging in age from birth to 84 years. Their conclusions are that both age-period changes and arteriosclerotic changes are maximal in the coronary arteries and minimal in the radial, and more or less intermediate in other vessels, such as the cerebral, splenic and renal arteries. They found that arteriosclerosis in the radial artery is such a rarity as to be negligible, and consequently the anatomical condition of the radial artery has no bearing on the degree of sclerosis in the visceral vessels.

E. S. MILLS

Hygiene and Public Health

The Physiological Response of Peritoneal Tissue to Certain Industrial and Pure Mineral Dusts. Miller, J. W. and Sayers, R. R., *Public Health Reports*, 1936, **51**: 1677.

The behaviour of certain dusts introduced into the peritoneal cavity has been described by the authors in earlier publications. The conclusions reached by them were that "live animal tissues in all parts of the body tend to react in essentially the same manner to foreign bodies, and that fibrous tissue is formed in the peritoneal cavity by quartz and is not formed by limestone and coal". The present study is a continuation of the earlier ones and confirms the conclusions reached. Briefly, suspensions of various dusts are injected into the peritoneal cavity of guinea pigs, and after intervals of 14, 30, 60 and 90 days the animals are killed and the peritoneal reactions studied.

The reactions fall into 3 groups: (1) a group in which the dust is absorbed without visible gross damage; (2) a group in which the dust initiated cellular proliferation followed by fibrosis and retrograde changes; (3) a group in which the dust remained inert in the tissues, neither being absorbed nor causing gross proliferation. In the first group fall the limestone and other calcium dusts, including Portland cement, which is largely calcium, although it does contain about 20 per cent silica. In the second group fall the dusts which are high in free silica, such as quartz. In the third group are a number of dusts; coal, talc, asbestos, soapstone, silicon carbide, sericite, etc.

The authors suggest that dusts of the first type might be considered relatively harmless, dusts of the second type, definitely dangerous, from their ability to produce fibrosis. The significance of dusts of the third type, which produce an inert reaction in the peritoneal cavity, is difficult to interpret, but their tendency to remain in the tissues should be considered as potentially harmful.

FRANK G. PEDLEY

The Causation of Pneumoconiosis. Drinker, P., *J. Ind. Hyg. & Toxicol.*, 1936, **18**: 524.

Drinker reviews succinctly the current views on the etiology of pneumoconiosis. Practically only two forms are of importance—silicosis and asbestosis. The proportion of Si O_2 is one controlling factor, *i.e.*, the higher the proportion, the greater the hazard; and the size of the particles, another; particles of about 3 microns or less in diameter are probably the active ones pathologically. Emphasis is laid on the value of dust samples and in this connection warning is given of the danger of assuming that dust collected from rafters and other places where it settles is typical of the dust inhaled by workers.

For practical purposes safe concentrations of pure quartz dust are less than 5 million per c.ft. For low quartz concentrations the permissible concentration may reach 50 million particles per c.ft. A statement of the Miners' Phthisis Medical Bureau (South Africa) in 1934 is quoted: "No 'New Rand Miner' who has entered the industry since August, 1923, *i.e.*, 10½ years ago, has as yet contracted silicosis".

FRANK G. PEDLEY

Obituaries

Since the reference in our last issue we have received several appreciative notices of the late Dr. D. A. Stewart which we readily print as being much more adequate. [ED.]

Dr. Stewart was born at Fletcher, Keith County, Ont. The lad was educated at Chatham, and when the family moved to Manitoba he took his Arts course at Manitoba College, graduating in 1899. Theology then claimed him, but while on a mission field in Frank, Alberta, his voice failed, and the example of the doctor in caring for the wounded after the disastrous rock slide induced him to enter medicine. In 1906 he graduated from Manitoba Medical College, having paid

The late David Alexander Stewart

his way by working as a reporter with the *Winnipeg Free Press*. For two years he served as an intern at the Winnipeg General Hospital, then became Field Secretary of the Anti-tuberculosis League of Manitoba, traversing the province to spread the gospel of the curability of tuberculosis and to raise funds for a sanatorium. His fiery zeal led to a breakdown, where it was discovered that his lungs were affected. A year at Trudeau Sanatorium, Saranac Lake, N.Y. and as resident in a Connecticut institution gave him first hand knowledge of sanatorium life, and so renewed him that he was able to work with his former vigour. Pelican Lake, near Ninette, was chosen as the site of the new Manitoba Sanatorium, and the first buildings were erected in 1910 with 60 beds available for patients.

Remarkable as were Dr. Stewart's activities in connection with tuberculosis, they did not limit the range of his interests nor absorb the whole of his dynamic energy. Medical history and the history of the fur-traders in the Canadian North-West found him an authority, and one of his special hobbies was to trace the sites of the forts of the Hudson's Bay, North-West, and the XYZ Companies along the Assiniboine and Souris rivers. For two years he was President of the Manitoba Historical and Scientific Society, and he was also a member of the Historic Sites Commission. His enthusiasm for education, beginning first with the sanatorium school and extending to the university and to adult education, led to an honorary LL.D. degree from Manitoba, the Honorary Presidency of the Manitoba Adult Education Committee, and to his election by the Alumni as a Governor of the University. In 1928 he was President of the Manitoba Medical Association, and in 1935 he was appointed Chairman of the Committee on Credentials and Ethics of the Canadian Medical Association, an office which he held up till his death. In this capacity he wrote a new Code, agreeable to present day conditions and graced with quotations from the great medical classics. It was characteristic of his devotion to duty that as late as four days before his death he supervised a letter to be sent to members of the Committee, and made marginal notes in his own handwriting.

His published work consisted of numerous papers reflecting the wide range of his interests, and marked with vigour and charm of style. He preached a wholesome and heartening philosophy of the sick room.

Though he had received no instruction in art, he did creditable work in dry point and water colours. At a recent exhibition of the Winnipeg Sketch Club four of his pictures were shown, one of them a view from his hospital window of the city atmosphere at 40 degrees below zero.

From its inception, the sanatorium was linked with the Medical School for the teaching of physical diagnosis. Not the least of Stewart's work was the training of young doctors in tuberculosis, and many who now occupy prominent posts will admit their obligation to his inspiration.

In 1915 Dr. Stewart married Ida K. Bradshaw, a graduate of the Winnipeg General Hospital School of Nursing, and one of the earliest Social Service nurses in Canada. Later she became widely known through her writings in the cause of peace. Delicate in health for long years, she moved to Winnipeg last autumn to be near her husband, but the change was too great for her strength, and she succumbed in November, only a few weeks before him.

"They were lovely and pleasant in their lives,
And in death they were not divided."

An only son, David Bradshaw Stewart, is a student in Medicine,

Dr. Stewart exhibited the "angelic conjunction" of physician and priest. All his work was permeated with a deep moral earnestness and he lived "as ever in the great taskmaster's eye".

ROSS MITCHELL

The passing of Dr. Stewart is felt as a personal loss by every one of the tuberculosis workers in Canada. His brilliant mind and forceful personality will be sorely missed in the councils of tuberculosis organizations. The high esteem in which he was held by his confrères was shown by his preferment to the highest positions of responsibility and honour. As leader of anti-tuberculosis work in Manitoba he lived to see a splendid organization developed and the death rate from tuberculosis reduced to one-fifth of what it had been. The loss of his stimulating personality and his aggressive leadership will be felt by every group of tuberculosis workers across Canada, but perhaps the greatest loss of all is his literary skill in interpreting clearly and forcibly the anti-tuberculosis movement to the masses of the people.

R. G. FERGUSON,
President, Canadian Tuberculosis Association.
Fort San, Sask.

David Stewart was much more than a man in a million. There are about ten million people in Canada but there are not nine other David Stewarts. To know him was a liberal education as well as an inspiration. His mind rose above other men's as his body did. He was alert to what he could do for people rather than what he could get from them, and though he was a dreamer he made most of his dreams come true. His mind was never idle; a sound physician, he was as well

a sociologist, an organizer, a bibliophile, an historian, an antiquarian, a writer of great ability, an artist, a charming talker, and, above all, a Christian gentleman.

In his versatility and insight he was the Canadian most like Osler, and about him there was an ectoplasm which suggested Robert Louis Stevenson.

I have just come across a letter from him dated 1928 from which I quote this paragraph.

"I was sorry to have the talk about putting some sick people to work cut short. The big need is the moral need to keep men from deteriorating in the region of the back bone and in its functions while a patch is being put upon lungs. It's wonderful how well the plan works and how easily when it gets a good start. In some groups it almost carries itself, some being teachers or encouragers to others.

"I am enclosing some mention of it in reprints but think I'll look for time to write up more definitely 'The Schoolmaster in the Hospital'."

The letter from which this was taken was written from a sick-bed.

In 1933 he gave an address entitled "Dickens the Reformer" which reveals two people, one was Charles Dickens and the other unconsciously was David Stewart, whose ambition was to

"Ring out the darkness of the land,
Ring in the Christ that is to be."

David Stewart made this country richer by his living and poorer by his dying. A. H. GORDON

Dr. Joseph Edgar Bates, of Toronto, died on February 21, 1937. He was born in 1895 and a graduate of the University of Toronto (1922).

Dr. Edwin Charles Beer, a member of the surgical staff of St. Michael's Hospital, Toronto, died on March 3, 1937. He was a graduate of Trinity University, Toronto (1903).

Dr. Wilfrid Bonnier, of Montreal, formerly Statistician of the Province of Quebec Health Department, died on February 12, 1937, aged sixty-five. He was a graduate of Laval University Medical School, Montreal (1899).

Dr. Elphege A. René de Cotret, well-known in the medical profession as an obstetrician and professor of the University of Montreal, died on February 7, 1937.

Dr. de Cotret was born in Montreal on October 6, 1861, the son of the late L. D. René de Cotret, lawyer, and of Emélie Pinet. He studied classics at the Collège de Montréal and entered the then Laval University of Montreal, to study medicine. After his graduation in 1888 he practised for a short time at St. Césaire before establishing himself in Montreal.

Several years later he was appointed assistant superintendent of the Hôpital de la Maternité and later became the superintendent of that institution after the death of Dr. Dagenais. In 1896 he was appointed Associate Professor of Obstetrics of Laval University. With the passing of years, appointments followed at Notre Dame Hospital and Laval University, of which he became one of the directors.

Dr. René de Cotret was also an author, having contributed "L'amour ne Meurt pas", "Les Voies de l'Amour", "Sœur ou Fiancée", "Suites de Couches Normales et Pathologiques", and "L'Obstetriques des Gardes Malades".

In 1888 he married Rose Alinda Content, who predeceased him in 1929. He is survived by a daughter, Mrs. Léon Lajoie and three brothers.

Dr. Agnes Douglas Craine, of Smith's Falls, Ont., died on February 26, 1937.

Doctor Craine was born at Smith's Falls on October 9, 1861, and was educated at the public and high schools of that place. She taught school for a few years and then entered Queen's University, graduating in medicine in 1888. She then took post-graduate work in Britain and obtained the Triple Qualification of Edinburgh and Glasgow. The rest of her life was spent in her home town.

Doctor Craine was the last of her family. She left the sum of $380,000 to Queen's University in memory of her family and as a tribute to the late Very Reverend George Monroe Grant, Principal of the University, who had opened the door to women for the study of medicine.

Dr. Andrew Hamilton Miller, of Hamilton, Ont., died on February 10, 1937, in his eighty-ninth year. He was a graduate of Trinity University (1877).

Dr. Joseph Alfred Montreuil, of Prince Albert, Sask., died on March 1, 1937.

Dr. Montreuil was born in Quebec in 1855, received his medical degree from Laval University, Quebec (1910), was an intern for a time in a Quebec hospital, and then continued his studies in Paris. He went to Prince Albert in 1912 and immediately begun the practice which he carried on until his death. Surviving are, his widow, one daughter Paule, and two sons Jilles and Marcel. His mother, one brother and five sisters, all of Quebec, also survive.

Dr. Richard James Shute, of Windsor, Ont., died on February 23, 1937, aged sixty-four years.

Dr. Shute was born in Grey County, near Owen Sound, in 1873, and was a graduate of the University of Western Ontario (1912). He is survived by his widow; his mother, Mrs. James Shute, Wiarton; three sons, Dr. Evan V. Shute, University of Western Ontario; Dr. Wilfrid E. Shute, senior in surgery at St. Michael's Hospital, Toronto, and Dr. Wallace Shute, Harper Hospital, Detroit.

Dr. John Addy Sponagle, of Middleton, N.S., died on February 19, 1937, aged seventy-six.

Dr. Sponagle was born in Parrsboro, N.S., and graduated from Dalhousie University, Halifax, in 1883. He went overseas with the 25th Battalion as a colonel in the Canadian Army Medical Corps and served in England and France during the Great War. Upon his return to Halifax in 1918, he was placed in charge of Camp Hill Military Hospital there, a position which he held for several years before retiring to Middleton.

Mayor of Middleton for two terms, Dr. Sponagle was a diligent worker for the town and prime mover for the newly-finished addition to the Soldier's Memorial Hospital there.

He is survived by two daughters, Mrs. H. B. Reach and Mrs. J. A. Reach.

Dr. Francis George Wallbridge, of Belleville, Ont., Chief Coroner for Hastings County, died on March 4, 1937. He had been in retirement for some years.

Dr. Wallbridge practised for twenty-two years at Midland before coming to Belleville seventeen years ago. In 1915 he went overseas as medical officer for the 157th Battalion, returning to Belleville after the war. His appointment as chief coroner came in 1935.

Surviving are his wife, formerly Charlotte Brown Keating; and one daughter, Mrs. Joseph Jeffrey, of London, Ont.

News Items

Great Britain

Reception at the Royal Society of Medicine

I am instructed by the Council of the Royal Society of Medicine to ask you to be so kind as to draw the attention of your readers to a Reception which the Royal Society of Medicine is giving on May 10th next at 8.30 p.m. in order to celebrate Coronation Week. It is the desire of my Council to entertain on this occasion medical men from the British Empire from overseas, whether Fellows of the Society or not, who will be in London on the occasion of the Coronation. As our accommodation is limited it would be of assistance if medical men wishing to avail themselves of the Society's hospitality on this occasion would communicate with me not later than May 1st.

Yours faithfully,
G. R. EDWARDS,
Secretary.

1 Wimpole St., London, W.1.
19th February, 1937.

Addendum to the B.P.

The British Drug Houses Ltd. has informed us that they have issued a Guide to the British Pharmacopœia. The firm will be glad to send a copy of this to members of the medical profession who will apply to them, Graham Street, City Road, London, England.

Alberta

Group hospitalization, a system by which a certain percentage of employees in any industry may provide for thirty days' hospitalization per year by paying sixty cents per month, collected by the group and sent to the central committee, is spreading. Its first real start was among certain citizens of Edmonton, but it is spreading into the rural communities where church, private or town hospitals exist. At the present time rural municipal hospitals charge one dollar per day, public rates for their taxpayers, and make up their operation deficits through taxation, but other hospitals have no such power, and consequently are adopting the group system.

The American College of Surgeons is holding a sectional meeting in Edmonton, March 24 and 25, 1937, and has been generous enough to invite every physician in the province to attend without registration fee. An attendance of five hundred is anticipated. Thus it is expected to reach as many Albertans as our own annual provincial convention.

For several years the university has put on a "refresher" course, during the week of May 15th, at the time of the annual University of Alberta Convocation. But, in view of the fact of the meeting of the American College of Surgeons shortly before, it is considered doubtful whether the men outside of Edmonton would come back for the course so soon after attending the Convention. It is possible the "refresher course" will not be held this year.

The Alberta Association, in cooperation with the Associations of Saskatchewan and British Columbia, has arranged for its annual meeting September 8th, 9th and 10th in the City of Edmonton. It is expected that the President and General Secretary of the Canadian Medical Association, together with two clinicians from outside the province, will assist at the meeting. The first day will be set aside for a meeting of the Board of Representatives and a meeting of the College of Physicians and Surgeons. This latter is not a meeting of the Council but of every medical practitioner in the province, when any individual registrant is free to discuss any question relative to the practice of medicine. At this meeting the Registrar reviews the work of the Council during the year.

There is a tendency for contract practice to increase, and it looks as though steps need to be taken to have one body and one body alone make all contracts, so they may be sufficiently comprehensive and fair to the public and profession.

The annual banquet of the Calgary Medical Society was held at the Renfrew Club on February 9, 1937, and was presided over by Dr. A. J. Fisher. Dr. Irving Bell represented the Edmonton Academy of Medicine. The guest-speaker of the evening was the Venerable Archdeacon Swanson, who gave a humorous address on experiences in clerical work and some of the incidents connected with members of our profession.

G. E. LEARMONTH

British Columbia

For some weeks after the end of January when the Health Insurance Committee of the College of Physicians and Surgeons of British Columbia, speaking for the profession, announced its refusal to accept the plan which the Health Insurance Commission proposed putting into operation on March 1st, Health Insurance has been front page news almost continuously in the daily press. As the *Vancouver Daily Province* of February 2nd said, "The fate of health insurance in British Columbia has ceased to be a fight between the Health Insurance Commission and medical men of British Columbia, and has become a major problem of provincial politics. . . . It was clear that the government itself, not Dr. Allon Peebles and his commission, must take final responsibility for deciding whether the insurance program is to go on without the doctors, postponed, or drastically overhauled."

The solidarity of the medical profession on this question throughout the province revealed by the secret ballot on their stand has not only amazed the government and the public at large but has astounded the doctors themselves: 614 ballots were returned, representing 96 per cent of the profession, and only 13 favoured the Health Insurance plan. The reaction of various individuals and groups towards the medical men's attitude has been interesting to watch. Judging from what one hears and overhears, and from the correspondence in the daily papers, the public in general eulogizes the profession and appears to realize that the doctors have been actuated as much by a regard for the public welfare as for their own professional interests. With rare exceptions letters in the newspapers have been commendatory; the exceptions have emanated largely from the radical labour element.

As for the reaction of government officials, evidence of an element in the liberal party moving to postpone the program for further consideration developed early. It was amusing to note the promptitude of the government in blaming its intelligence service for failing to warn it of approaching danger from medical opposition. They appear to have felt that all was clear sailing ahead and that the attitude of the medical profession would be one of supine acquiescence. As feeling strengthened in government circles that the program would have to be postponed, it was freely predicted that Hon. Dr. Weir, Provincial Secretary and father of the Health Insurance Act, would resign if this happened, and further that the cabinet as a whole would prefer to accept this rather than inaugurate the scheme without the cooperation of the medical profession. At the same time the government was coming to a surprised realization that the public as a whole was unfavourable to the Act, and considering what the effect might be on the approaching

general election. In the meantime Dr. Weir kept silence until February 9th. On that day a luncheon meeting of the Vancouver Board of Trade was held under the auspices of the British Columbia Medical Association. The large crowd of business and professional men which packed the Aztec Room of the Hotel Georgia was addressed by Dr. Wallace Wilson, vice-chairman of the Health Insurance Committee of the College of Physicians and Surgeons, and listened to a full representation of the medical profession's attitude and its reasons for rejecting the Health Insurance scheme. By a special province-wide hook-up Dr. Wilson's address was broadcast to all parts of the province.

The daily press on that date also carried a statement prepared by Hon. Dr. Weir, attacking the stand of the doctors. He characterized the statements regarding the lack of provision for the indigents as "completely untrue". To support this he quoted figures stated to represent amounts spent in the last fiscal year by the province and municipalities on medical service to indigents, and concluded by remarking that he did not desire to comment on the present controversy between the Health Insurance Commission and the medical profession; that it was the responsibility of the commission to work out arrangements for doctors' services under the Act; and that the question of indigent medical services was not within the jurisdiction of the Commission. On the following day the cabinet announced that Health Insurance would be indefinitely postponed and its ultimate future would not be decided until after the return of the Premier, Hon. T. D. Pattullo, from Ottawa in March.

On February 15th, Dr. Weir returned to his office, convalescing from influenza, and declared "I would rather be defeated at the polls fighting for health insurance than be successful without it". At the same time he effectually quieted the rumours about his resignation by stating that he had no such intention. On the same night Dr. Peebles, chairman of the Commission, opened what was planned to be a series of radio speeches as part of a campaign to swing public favour to health insurance.

An amusing note was furnished about this time by the statement quoted in the *Vancouver Daily Province* of a young Vancouver barrister and former Rhodes scholar, in the course of an address to the Vancouver Centre Liberal Association, that he had estimated that a doctor who chose to take only health insurance patients could make $6,200 a year clear of expenses on a group of 1,500 people!

A dispatch from Toronto reported that Premier Pattullo in an interview there had said that health insurance would succeed in British Columbia as it had done in England. Referring to those such as farmers and indigents at present excluded from the scheme, he said that they would eventually be included, and meanwhile they were no worse off than before.

On February 16th Mr. Percy Bengough, addressing the Vancouver and New Westminster Trades and Labour Council, of which body he is secretary, made a bitter attack on the opponents of the Health Insurance scheme. Mr. Bengough is also one of the members of the Health Insurance Commission. Proceeding along the customary lines he endeavoured to show that the opposition was a class-warfare manifestation. Referring to the Canadian Manufacturers' Association, Shipping Federation of British Columbia, British Columbia Loggers' Association, Vancouver Merchants' Exchange and the Board of Trade and others reported as opposing the scheme, he said "Do you think it likely that these organizations are fighting the Act for the benefit of the workers?" He charged that "something like $50,000 has been subscribed to fight health insurance. I have heard that they have $100,000." Turning to the doctors, he said that if the Act was not adequate they were in the main to blame since most of the changes had been made at their instigation. The reason was, he said, that the doctors wanted a scheme to cover only those who cannot now afford to pay a doctor, and they would then be free to charge everyone else as they saw fit. Mr. Birt Showler, teamsters' delegate, who has been one of the writers of letters to the daily press supporting the scheme, said at the same meeting "if labour organizations attempted to do what doctors and others are doing the government would call out the militia". Dr. J. J. Gillis, Liberal M.L.A. of Merritt, British Columbia, has challenged Mr. Bengough to give his authority for the statements made concerning the fund subscribed to fight health insurance and intimidation of employees by these opponents. He believes Health Commissioner Bengough's statements do not reflect the opinion of labour, and enjoins him as a responsible person to refrain from repeating such wild statements. It is notable that the Victoria Trades and Labour Council did not approve the scheme.

Definite instructions to postpone operation of the Health Insurance Commission *sine die* were received from Premier Pattullo, absent in Ottawa, on February 19th. A plebiscite on the question is forecast by some political prophets, but in the meantime the staff of the Health Insurance Commission in Victoria has been reduced from some sixty or seventy to a skeleton formation.

In a Toronto dispatch to the *Vancouver Province*, captioned "Health Plan Draws Praise," Lieut.-Governor Herbert A. Bruce, M.D., of Ontario is quoted as saying with reference to the British Columbia Health Insurance Commission's Plan and the objections to it raised by the doctors, "it would be unfair to minimize these objections. They are real and honestly put forward. . . . All honour to British Columbia for its courage. Any health scheme of insurance, however, which excludes the lowest income groups and the indigents is evidently far from a perfect scheme and open to many objections."

The board of directors of the Nanaimo General Hospital are considering the advisability of adding a new wing at an approximate cost of $75,000. All four floors of the institution are in use, and the limit of 65 patients has forced an increase in the nursing staff, which had to be housed in the institution, there being inadequate accommodation in the nurses' home.

Under the plan for "Coronation Functions" being arranged by the United Commercial Travellers throughout the Dominion on April 14th, Vancouver's leading activity will be a formal banquet in the Hotel Vancouver at which Lieut.-Governor Eric Hamber and other members of the British Columbia Cancer Foundation will be hosts. The diners will pay $10.00 a plate. Other dinners and dances will also be held, and the proceeds will be given to the British Columbia Cancer Foundation.

At the twenty-fifth annual meeting of the Board of Directors of the Vancouver General Hospital held on February 10th, Mr. J. H. McVety, the honorary treasurer, refuted the charge that "poor people have difficulty in getting hospital service," which has been made on several occasions recently. He stated that free services given to indigents and others unable to pay were worth $778,640 during the past year. This amount was almost exactly equal to the combined grants from the Provincial Government, the City of Vancouver, and other contributing municipalities, and the operating deficit. Rev. C. C. Owen, the hospital host, said "Relief cases get as good treatment as any". D. N. Hossie, Esq., K.C., chairman of the Board of Directors, stated that the present need for enlarged accommodation at the hospital constituted a situation of "pressing urgency". Dr. A. K. Haywood, Superintendent, made the statement that plans for introducing a money by-law at the last civic election for a new nurses' home, although the need for it was urgent and serious, had to be dropped on account of the still more serious condition in the wards.

EMMENIN

at the menopause

The value of Emmenin in the treatment of menopausal symptoms has been ably demonstrated by many clinicians. Several papers have been published on this subject and it is particularly interesting to note that the most distressing symptoms of the menopause,—the hot flushes, mental irritability and physical weakness—usually show a gratifying response to Emmenin. The fact that Emmenin is orally-active constitutes a decided advantage where other than oral therapy might cause an unfavourable reaction.

EMMENIN

in dysmenorrhoea

In this condition, where treatment should be continued throughout several intermenstrual periods, convenience of administration assumes considerable importance. As in menopausal symptoms, the oral activity of Emmenin permits a routine that is simple for the patient to follow. In general, treatment of at least three months' duration is necessary to produce a permanent degree of relief.

EMMENIN

in menstrual migraine

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A delegation of doctors in Vernon, British Columbia, has appeared before the City Council requesting an arrangement whereby they will be paid for services rendered to indigents. As a basis for negotiation, their spokesman, Dr. J. E. Harvey, asked for a scale of $1.50 a call and $1.00 for an office consultation, with a reasonable fee for maternity cases. No decision was reached by the Council. The Jubilee Hospital in Vernon has reported the highest percentage of charges collected in any year in its history, the figure being 70 per cent of the charges levied. The creation of a hospital insurance scheme, which now covers 2,980 contract members and their dependents, is believed to have contributed very greatly to the successful financing of the institution.

D. E. H. CLEVELAND

Manitoba

The three-day post-graduate course in Obstetrics and Gynæcology, given by the Faculty of Medicine of the University of Manitoba on February 17th, 18th and 19th, was a marked success. It was attended by more than twenty-five rural practitioners.

At a meeting of the Manitoba Sanatorium Board held on March 2nd, Dr. E. L. Ross, who for several years has been Assistant Superintendent at Ninette Sanatorium, was appointed Superintendent to succeed the late Dr. D. A. Stewart.

One of the first acts of the Manitoba Legislative Assembly was to pass a resolution expressing the great loss suffered by the province in the death of Dr. D. A. Stewart, who for over twenty-five years had been Superintendent of the Manitoba Sanatorium.

The sixtieth anniversary of the founding of the University of Manitoba will be held at Convocation in May. It is rumoured that the honorary LL.D. degree will be conferred on one or two eminent medical men.

ROSS MITCHELL

New Brunswick

On March 4th the staff of the Chipman Memorial Hospital in St. Stephen, in conjunction with the Washington County Medical Society, entertained at a staff luncheon. Following this an address was presented by Dr. Charles F. Painter, of Boston, on the general subject of orthopædics. Physicians throughout the lower part of the province were invited to be present at this meeting.

Dr. R. J. Collins, Superintendent of the Saint John Tuberculosis Hospital, has been granted two months' leave of absence, to enable him to attend the conference on the After-Care of Tuberculosis, which is to be held in London, May 3rd to 13th. This conference has been arranged by the Over-Seas League, and is under the auspices of Sir Varrier Jones, head of the Papworth Colony for the after-care of tuberculosis.

The Public Health Authorities in the Province of New Brunswick are keeping close watch on the prevalence of influenza. Their reports indicate a considerable number of patients suffering from influenza at this time. There is, however, a very much larger number ill throughout the province whose cases do not come to the attention of a physician, so it appears that influenza at present is almost epidemic. A large number of physicians have been attacked by the disease. Mumps and measles are also fairly prevalent, and again a fair number of physicians have been the victim of mumps. Hospitals throughout the province report that their wards have been filled to capacity for some time past.

Dr. Norman Skinner has begun practice in the City of Saint John.

A. STANLEY KIRKLAND

Nova Scotia

At the annual meeting of the Halifax Visiting Dispensary Mr. E. L. Miller was elected president, in succession to Mr. C. H. Mitchell who died last year. A letter from the Council of Social Agencies was read, requesting a twenty-four hour and a week-end medical service for the poor of Halifax. This item was discussed in detail, but it was not approved as the money for such a service is not available. The report of the secretary pointed out that the past year was a healthier one, particularly in epidemic ailments. It stressed the value of the immunization work carried on by the Dalhousie Health Clinic. More prescriptions were dispensed than in the previous year.

The air-port buildings at Upper North Sydney, unoccupied for many years, were inspected recently by a committee from the Cape Breton Joint Expenditure Board and were approved as suitable for an additional institution for the insane. At present a total of about $45,000 is spent annually by the Board to care for patients at Halifax and Mabou. Concentration of these cases in Cape Breton would prove more economical. It is proposed to invite a representative from the Provincial Health Department to inspect the buildings.

The Glace Bay Branch of the Canadian Legion has endorsed as a "temporary measure" the Provincial Government's recommendation to erect, equip and supervise the tuberculosis annexes in connection with the two hospitals of Glace Bay. It will be recalled that a considerable amount of opinion favours the erection of a central institution for tuberculosis in Cape Breton, but the report of the physicians who made the survey favoured the erection of two annexes.

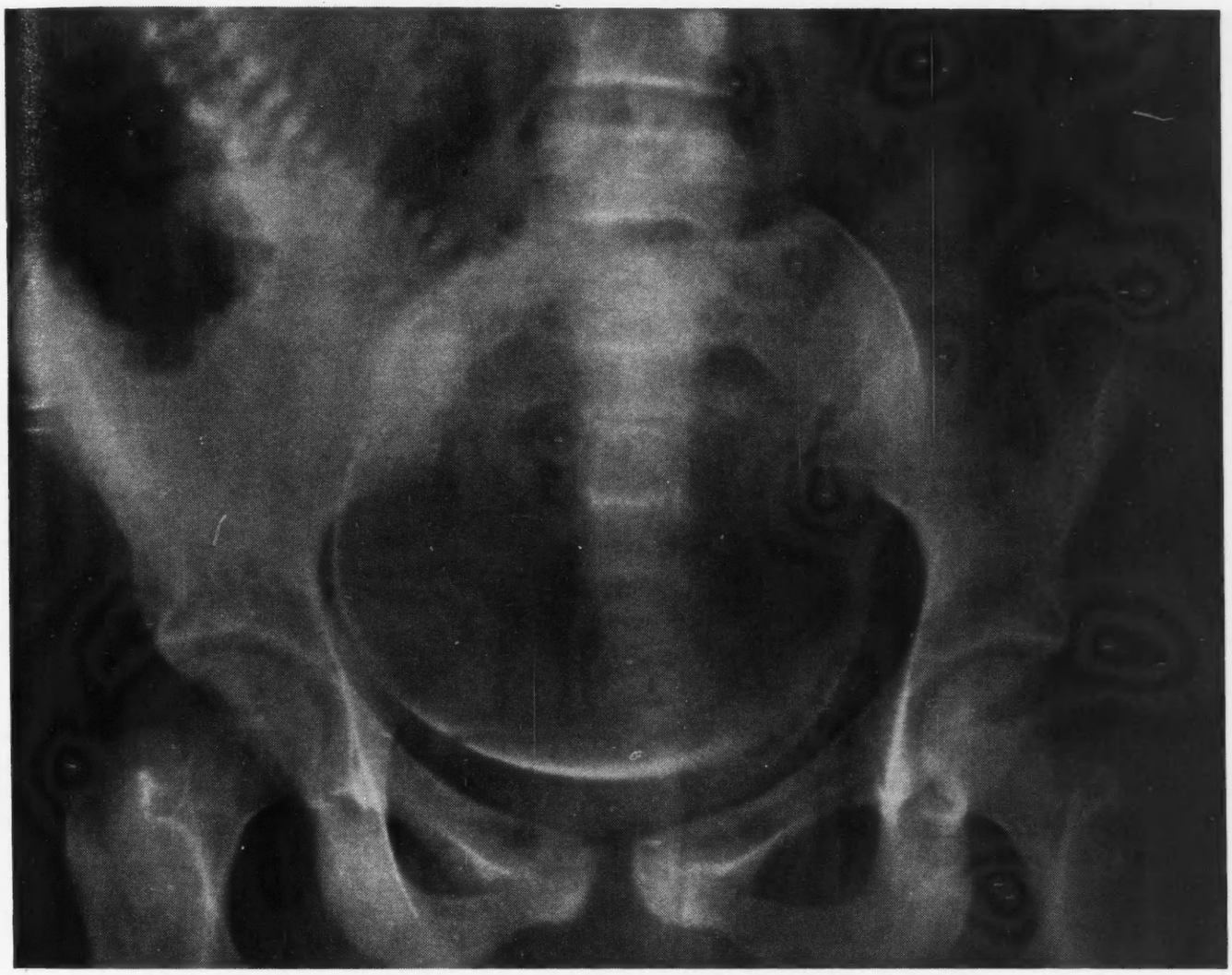
The annual report of the medical officer of health, Dr. W. D. Forrest, and of the Warden of Halifax County urges the need of active measures to combat the spread of tuberculosis in the county. The situation is fast becoming worse, for the number of cases far exceeded those of previous years. Dr. Forrest urged the appointment of a committee to interview authorities of the Provincial Health Department to work out a scheme to prevent the spread of this disease. The report further states that there were no cases of typhoid fever in the county during the year.

N. B. DREYER

Ontario

The Hospital for Sick Children, Toronto. — On January 23rd, the Department of Psychological Medicine of the Hospital for Sick Children and the Department of Pædiatrics, University of Toronto, was formally opened by His Honour the Lieut.-Governor of Ontario, Dr. H. A. Bruce. Dr. Alan Brown was Chairman of the meeting, and Dr. C. M. Hincks, Chairman of the National Committee on Mental Hygiene, introduced the speakers, Dr. Edwards A. Park, Professor of Pædiatrics and Dr. Leo Kanner, Associate Professor of Psychiatry, Johns Hopkins University. Both speakers stressed the importance of liaison and cooperation between the pædiatrician and the psychiatrist.

The importance of the opening of such a clinic, which is the first of its kind in Canada, and the fourth on this continent, was evinced by the great interest shown by those engaged in this type of work. Of the 175 invitations sent out, 157 were accepted, and guests were present from Montreal, Ottawa, and various points in Ontario. Dr. Charles Bradley, of East Providence,



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RADIOGRAPHS PROVIDE DIAGNOSTIC FACTS

R.I., Superintendent of the Emma Pendleton Bradley Home, was also present.

President Cody congratulated the hospital on its excellent work among the children of the city and of the province, and stressed the importance of this step towards the improvement and maintenance of the mental health of the child.

Among those present were Mrs. Bruce, Mrs. Cody, Sir Frederick Banting, Dr. W. E. Gallie, Dr. George Boyer, Mr. R. A. Laidlaw and Dr. B. T. McGhie.

The clinic is in a separate building situated on hospital property behind the Nurses' Residence and the rooms have been arranged to give as pleasant and homelike an atmosphere as possible. The clinic will be devoted to the mental health of childhood and will attempt to solve the various problems of that nature, whether they be slight deviations from normal, or marked ones involving both the child and his environment. In the treatment of these problems both individual psychotherapy and a generous use of the community resources will be employed. The staff will be under the direction of Dr. George Boyer, who has been chief of the Neurological Service since its inception in 1919, and will consist of two psychiatrists, Dr. Wm. A. Hawke and Dr. J. D. Griffin, a social worker, Miss Hally, and several psychometrists.

It is the heartfelt wish that through this early work with the individual the clinic may be able to decrease the incidence of mental disease in adult life, to lower delinquency and similar anti-social problems in the community, and to also serve as a centre for the dissemination of knowledge to both medical students and practitioners.

The many friends of the late Dr. Peter McGibbon, of Bracebridge, for many years Member of Parliament for Muskoka, will be pleased to know that the town of Bracebridge is making good progress in its effort to raise a fund of $20,000, to be expended on the Bracebridge Hospital as a memorial to Dr. McGibbon's work in his own community. Subscriptions have been received from many parts of Canada. The lists are still open. The amount of money already in sight is sufficient to provide a much needed waiting-room, office quarters, and elevator, and will probably include an attractive memorial entrance.

A deputation from the City Council and Hospital Board is seeking approval of a 54-bed extension to the Brantford General Hospital, at a cost of $100,000.

Dr. Charles D. Parfitt, since 1901 associated with tuberculosis work in Ontario, consultant to the Department of Soldiers' Civil Re-establishment, and until recently the Director of Calydor Sanatorium in Muskoka, has accepted a position as Superintendent and Medical Director of the Lumis Sanatorium at Liberty in New York State. The Laennec Society of Ontario honoured Dr. Parfitt by a farewell dinner at the York Club on the evening of Wednesday, February 24th. General regret was expressed by his many friends in the profession and many former patients at his departure. Dr. Parfitt's leaving will be a great loss to Canadian medicine.

Dr. D. O. Ferris, of Niagara Falls, Ont., Secretary of the Niagara Falls Medical Society, has been appointed to the Council of Queen's University.

A diploma of Fellowship of the Royal College of Surgeons has been granted to Russell Kneale Magee, of the University of Toronto (B.A. 1927, M.D. 1930) and St. Bartholomew's Hospital. A diploma of Membership of the Royal College of Physicians has been granted to Francis Staples Brien, of the University of Toronto (M.B. 1933), and to John Clifford Richardson, of the University of Toronto (M.D. 1932). J. H. ELLIOTT

Quebec

Dr. F. E. McKenty has been appointed to succeed the late Dr. Scrimger as Surgeon-in-Chief of the Royal Victoria Hospital, Montreal.

After graduating at McGill University (1904) Dr. McKenty went to Freiburg, to study pathology under Professor Aschoff. He then went to Berlin where he remained a year studying surgery under Professor Bier and pathology under Professor Pick. After continuing his studies in Vienna and Paris, he returned to America and visited the various clinics in the United States. He began his practice in Montreal in 1909 the first years of which were spent mostly in research and laboratory work.

In 1909 he was given a position as clinical assistant in surgery at the Royal Victoria Hospital, and was the first to introduce spinal anæsthesia into the Royal Victoria Hospital, having had considerable experience with it while working under Professor Bier in Berlin.

He has contributed papers on blastomycosis, actinomycosis, tumours of the appendix, Paget's disease of the breast, the embryology of the neck and tumours of the neck, and on the treatment of surgical tuberculosis with reference to tuberculin. In 1909 also, he was appointed demonstrator in anatomy at McGill, which position he held for five years. He also lectured during the same period in operative surgery and applied anatomy.

During the war, after spending some time in England, he went to France and served in the base hospital at Tréport five months, and with the McGill unit at Boulogne for six months. On his return from the war, he was appointed associate in surgery; a few years later he became assistant surgeon; finally, three years ago, he was appointed surgeon. In all these several positions he was actively engaged in teaching clinical surgery, and during the past four years he has controlled all the fourth year teaching in the Royal Victoria Hospital.

Dr. McKenty is an F.R.C.S.(Eng.) and also a Fellow of both the American and Canadian Colleges of Surgeons. Last year, he became assistant professor of surgery at McGill.

Dr. Albert Lesage, of the University of Montreal, was elected a corresponding member of the French Academy of Medicine on February 16th. He received 65 votes, an unusually high number. He was sponsored by Professor Emile Sergent.

Dr. Lesage is one of Canada's foremost French-Canadian doctors. From 1910 to 1927 he was professor of internal pathology at the faculty of medicine of the University of Montreal. Since 1927 he has been professor of clinical medicine at Notre Dame Hospital.

He was the first secretary of the Association of French-speaking Physicians of North America, and was its president from 1926 to 1928. From 1925 to 1932 he served as a director of the Association of French-speaking Physicians (France), an honour shared by few other Canadian doctors.

Dr. Lesage has been editor-in-chief of our esteemed contemporary, *l'Union Médicale du Canada*, the chief French medical journal in North America, since 1900. He is a corresponding member of the Medical Society of Paris Hospitals. He is also a Fellow of the Royal College of Physicians.

Several times Dr. Lesage has represented the Dominion and the Quebec governments at international congresses, namely in 1925, at Nancy, France, at the convention of French-speaking doctors; at the same meeting in Paris, last autumn; and at the meeting of the Latin medical press, in Venice, last September.

Dr. Lesage has been honoured many times by the Government of the French Republic. In 1900 he was made an Officer d'Académie, four years later he was awarded the title of Officer of Public Instruction, and in 1927 he was created a Knight of the Legion of Honour.

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He was born in St. Raymond, Portneuf county, in 1869, and studied at the Quebec Seminary and Laval University, Montreal. Afterward he did post-graduate work at the University of Paris and obtained the degree of Doctor in Medicine.

Dr. J. C. Meakins, physician-in-chief at the Royal Victoria Hospital and head of the department of medicine at McGill University, left Montreal on March 24th for Australia, where he has been invited by the post-graduate committee in medicine, University of Sydney, New South Wales, to deliver a series of lectures. He expects to return early in August.

Saskatchewan

Dr. George R. Walton, D.P.H., has been appointed medical officer of health of Regina, Sask. Dr. Walton, who served in the 58th Battalion overseas, graduated in medicine from the University of Toronto in 1923. After an internship in the Regina General Hospital in 1923 he practised with Dr. R. G. Scott at Wakaw. In 1927 he was appointed medical officer to the Department of Railways and Canals, Canada, and was in charge of the department's hospital and medical work at Churchill, Man., for six years. In 1933 he resigned this appointment to take the course leading to the Diploma in Public Health at the School of Hygiene, University of Toronto, and thereafter spent a year in research work in the School. Before taking up his duties in Regina he served as medical officer of health of Pelee Island, Ont.

LILLIAN A. CHASE

Book Reviews

Textbook of Medicine. By various authors. Edited by J. J. Conybeare, M.C., M.D., F.R.C.P., Assistant Physician to Guy's Hospital, London. Third edition, 1027 pages, illustrated. Price $6.25. E. & S. Livingstone, Edinburgh; Macmillan Co., Toronto, 1936.

The publication of a third edition within seven years indicates that this textbook has made a place for itself. With a slight increase in size there has been made a further reduction in the price. The section on renal diseases is completely new, and there is much that is new in the section on the anæmias; that on coronary occlusion is extended. Though most of the contributors are English physicians both India and South Africa are represented, and Trevor Owen, of Toronto, writes on disease of the liver, gall bladder, pancreas and peritoneum. The pages could well be enlarged to lessen the thickness of the book. The inner margin of each page is too narrow for comfortable reading.

Textbook of General Surgery. Warren H. Cole, M.D., F.A.C.S., Professor of Surgery, Illinois College of Medicine, and Robert Elman, M.D., Associate Professor of Surgery, Washington University School of Medicine, St. Louis. 1031 pages, illustrated. Price $10.00. D. Appleton-Century Co., New York, 1936.

The material from which this work is compiled was first gathered in lecture and clinical courses for junior students in the Washington School of Medicine. This was revised and enlarged during a period of eight years, so that the book represents the ripened experience of two successful clinicians. Physiological concepts have been emphasized and pathogenesis stressed, while detail and, especially, operative procedures have been largely omitted. Each of the thirty chapters ends with a bibliography of works of reference, which vastly increases the value of the book to senior students and practitioners. The sections on surgery of the nervous system of the chest and genito-urinary system are especially good. The discussion on fractures is, perhaps, somewhat sketchy, but sound principles are emphasized and details may be found in the special books recommended.

The text is written in a clear expository style and the matter is up to date. The reviewer takes exception to the credit given Péan as the inventor of pressure forceps. Spencer Wells has a good claim to the distinction, but his name is not mentioned.

Diseases of Infancy and Childhood. Wilfred Sheldon, M.D., F.R.C.P., Physician for Diseases of Children, King's College, etc. 738 pages, illustrated. Price 21s. J. & A. Churchill, London, 1936.

This is more than "just another" textbook on pædiatrics. The author is following in the footsteps of Dr. Still at King's College Hospital in London, where he is physician to the children's wards. His writing also appears to be following Dr. Still in that what he offers is written in a lucid, logical and scholarly manner, and obviously expresses the reasoned judgment of his own experience. A volume of only a little over 700 pages, it is most complete in its scope. The principles of diagnosis and treatment in clinical medicine are more strongly stressed than in many other similar works.

One chapter covers completely the long list of proprietary foods and their place in infant feeding. This is perhaps of little interest to practitioners on this side of the Atlantic ocean. Another chapter which is unusual and of value is that on "Nasal Accessory Sinus Disease" written by James Crooks.

This book is to be commended, and will be found most useful by both general practitioner and specialist.

Lectures on Embolism and other Surgical Subjects. The Abraham Flexner Lectures—Series Number Four. Gunnar Nyström, M.D., Professor of Surgery, University of Uppsala, Sweden. 213 pages, illustrated. Price $3.00. Williams and Wilkins, Baltimore, 1936.

It has recently become the custom to exchange students from one university to another, nurses from one hospital training school to one in a far distant country; and often clinical professors of surgery are exchanged for the purpose of giving a series of lectures on subjects in which they are experts. The coming to Vanderbilt University of Dr. Gunnar Nyström, Professor of Surgery, Chief of the Surgical Clinic and Director of the University Hospital, Uppsala, Sweden, to give the fourth series of the Abraham Flexner Lectures, is an outstanding example of the interchange of surgical brains and experience now the vogue in today's methods of medical education.

No better series of lectures is available in book form than is presented in this volume, and no more inclusive series of subjects of paramount interest could well be chosen than arterial embolism, appendicitis, cytology of joint exudates, and fractures of the neck of the femur.

Dr. Nyström wastes no time in discussing the literature or the experience of others, but brings to us the unique, extensive and highly successful methods of arterial surgery used in Sweden, where the second successful case of removal of a femoral arterial embolus was reported, and where Alexis Carrell in 1912 received from the King of Sweden the Nobel prize for his research on the suture of blood vessels and his work on the transplantation of vessels and organs. It is, therefore, most fitting that Sweden should send to America her foremost exponent of arterial surgery in northern Europe. No surgeon can do better than provide himself with this volume if he expects to undertake embolectomy to save life and limb.

In Sweden the hospitals are able to furnish accurate figures, and his summary of appendicitis from the first appendectomy in Uppsala in 1888, by Lennander, to the end of 1934 is well worth reading. The increase in

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Prices and information relating to the use of these adrenal-gland products will be supplied gladly upon request.

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America in the death rate from appendicitis has been commented on widely here; and the average mortality in Swedish hospitals from 1913 to 1931, shows a similar rise, though the death rate following operation in acute appendicitis appears to have fallen to between 2 and 2½ per cent in Sweden in the years 1922 to 1934.

Dr. Nyström's personal study of the cytology of joint exudates includes, of course, a classification of joint diseases, with a subdivision of toxic synovitis, which offers a field for controversial discussion, but his illustrations of coloured smears show a method employed by him for diagnosis and the selection of appropriate treatment which is well worthy of trial. He sums up in seven conclusions the results of his studies, and the cytological findings are concisely expressed in the seventh, "The cytological picture of the joint exudate depends more upon the intensity than upon the origin of the morbid process". No surgeon who is up-to-date in his treatment of fractures of the "surgical" neck of the femur can fail to be interested in and encouraged by Dr. Nyström's presentation of Sven Johansson's method of treatment in Gothenburg.

The book is priced at three dollars, but you will not want your money back!

Textbook of Pathology. Sir Robert Muir, M.A., M.D., Sc.D., LL.D., F.R.S., Professor of Pathology, University of Glasgow, etc. Fourth edition, 994 pages, illustrated. Price $10.50. Ed. Arnold, London; Macmillan Co., Toronto, 1936.

This work is primarily intended for students and first appeared in 1924. It has run into four editions and has, in addition, been reprinted five times. There can, therefore, be no doubt as to its popularity. Indeed, even a casual glance into its pages dealing with any of the live topics of the day will reveal a masterly grasp of the subject, yet that topic is dealt with concisely, and judicially. In a few words the reader is put *au fait* with the facts and possible trends, and the author frequently gives his own opinion. From the standpoint of the learner this is admirable. The work follows in plan the well-beaten path which is familiar to the older generation of pathologists, but it is doubtful if there is any better way, at least for teaching purposes. In every case the latest investigations are referred to and the lesions of disease are related to their clinical manifestations. Much of the theory and debatable matter is proffered in small type, and the result is that we get a large amount of up-to-date information in a reasonable space. The book, while comprehensive, is of manageable size. It is written by a master in his subject and can be highly recommended as entirely adequate for its purpose. It is not hard to find reasons for its popularity. The student could have no better guide.

The Principles of Bacteriology and Immunity. W. W. C. Topley, M.A., M.D., M.Sc., F.R.C.P., F.R.S., and G. S. Wilson, M.D., F.R.C.S., D.P.H. Second edition, 1645 pages, illustrated. Price $15.00. Ed. Arnold, London; Macmillan Co., Toronto, 1936.

The first edition of this excellent work was published in 1929 and was enthusiastically received. The new volume is an undoubted success, as the authors have spared no effort in bringing the material up to date. The addition of reviews and comments on recent important work has greatly increased its value. In response to many requests the authors have combined the work into one volume. While there is some advantage in the change, the book, consisting of 1645 pages, is somewhat awkward to handle. (The two volumes of the first addition totalled 1300 pages). The arrangement is the same, except that in the systematic description of bacteria a fuller account of their pathogenicity to animals and man is included.

The work shows how widely the authors have been in touch with every phase of progress in bacteriology. Each chapter is well illustrated, contains many tables and figures, and ends with an extensive bibliography. The book is remarkably well written and has no rival. It is to be recommended not only to the medical bacteriologist, research worker, and post-graduate in bacteriology but also to the internist, to whom it should prove an excellent aid in clinical diagnosis. (Our review of the first edition can be found in the *Journal*, 1930, **23**: 126).

The Digestive Tract. A radiological study of its anatomy, physiology and pathology. Alfred E. Barclay, O.B.E., M.A., M.D., D.M.R. & E., M.R.C.P., F.A.C.R., Honorary Radiologist to Nuffield Institute for Medical Research, Oxford. Second edition, 427 pages, illustrated. Price $10.75. The Cambridge University Press; Macmillan, Toronto, 1936.

This book is a very complete treatise covering the x-ray examination of the gastro-intestinal tract. Methods of examination are detailed, information for the novice, and refinements of technique for the expert. Clear reproduction of x-ray film findings exemplify the normal, contrasted with examples of various common and rare pathological conditions. All the chapters are arranged in a most logical order and bear out the author's emphasis laid on the absolute necessity of a fixed routine, which must be followed if a maximum of information is to be obtained. It is evident that this work is not merely a compilation of this author's own researches and observations, but also contains contributions from his many radiological friends the world over, evidenced particularly by the inclusion of illustrations supplied by continental, American, Canadian, and British roentgenologists. Special chapters on the staffing of an x-ray department, x-ray records, ownership of films, etc., add to the usefulness of the book as a hospital reference volume, while the discussion of x-ray protective measures and roentgenological risks is of more particular interest to the members of the specialty.

Written by an acknowledged master of his craft and produced in a good binding on excellent paper, the book will find ready acceptance by all English-speaking roentgenologists.

Illustrations of Regional Anatomy. E. B. Jamieson, M.D., Senior Demonstrator, Anatomy Department, University of Edinburgh. Section VI; Upper Limb, 42 plates, price $2.25. Section VII; Lower Limb, 52 plates, price $3.00. E. & S. Livingstone, Edinburgh; Macmillan Co., Toronto, 1936.

In this *Journal* (May, 1935, p. 604) we reviewed favourably Sections I to V of this series. With Sections VI and VII, on the limbs, the series is now complete. The plates are made from drawings of bones and from actual dissections. Prepared primarily for students, these excellent colour reproductions should serve a useful purpose for the surgeon and teacher.

Practical Examination of Personality and Behaviour Disorders, Adults and Children. Kenneth E. Appel, M.D., Ph.D., Sc.D., Assistant Professor of Psychiatry, and Edward A. Strecker, M.D., A.M., Sc.D., Professor of Psychiatry, University of Pennsylvania. 219 pages. Price $2.00. Macmillan Co., New York and Toronto, 1936.

In this book the authors present a detailed summary of the present-day psychiatric examination methods for adults and children. A chapter on the art of psychiatric examination deals with the fundamental essentials in the physician-patient relationship and analyzes the various aspects of the art of medical practice in general, with particular emphasis on the manner in which successful rapport may be established between the physician and the patient. Detailed outlines for psychiatric examination of various types of patients then follow, and the book is concluded with a series of very helpful sugges-

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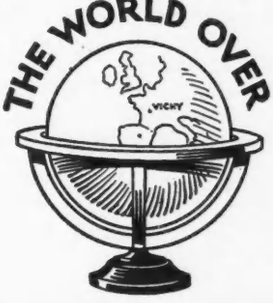

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tions of a practical nature for coping with such children's problems as temper tantrums, enuresis, jealousy, fears and other serious behaviour problems.

The clear, concise style which the authors employ throughout the book, and particularly the numerous illustrations used to indicate the various phases of the psychiatric examination procedure, should make this book a very real source of help to the profession at large.

The Medical Dictator and other Biographical Studies. Major Greenwood, F.R.S., D.Sc., F.R.C.P. 213 pages. Price 7s. 6d. net. Williams & Norgate, Ltd., London, 1936.

By the "Medical Dictator" Professor Greenwood means Galen, who, "having in life no more importance than a successful physician is likely to have, after his death acquired authority over members of his profession which was unlimited for more than thirteen hundred years. . . ." Galen is now only a name which to most of us has only the haziest of associations. Professor Greenwood's essay on him should help to correct this. He tries to show us why we should examine Galen. Why was he so powerful an influence in medicine? Is it not possible that he is yet exerting an influence, even if this is not consciously realized. Few living medical writers are so well qualified as the author of these essays to reanimate such a subject. He has style, and he knows what Galen's books contain.

There are other essays in this collection, however, and that which will probably be more immediately interesting to a Canadian audience is the one on Osler. Here again the author looks at his subject freshly. In the case of Galen his aim was to restore to a great figure something of a lustre which has grown dim. In the case of Osler he would moderate the hero-worship which so far has been so undiscriminating. It requires energetic measures however to shake off the spell of Sir William's personality, and to write with the cold detachment that a statistician of Professor Greenwood's standing must maintain. His opening sentence is an example of this not entirely successful attempt: "To the few medical men addicted to arithmetical statements, and to the many who care for what old-fashioned people call good men and younger people, white men, the life of William Osler will always be interesting." There is, no attempt at belittlement. But it is very difficult, even for a writer as able as Professor Greenwood, to reduce so vivid a personality to a well-proportioned sketch. We must await posterity's verdict before we can accept such a summing-up as: "I daresay that in a few generations Osler will be as soundly forgotten as Thomas Watson or even William Jenner, that he will be at most an occasionally cited name." We hope that Professor Greenwood will give us more essays.

Medical Classics. Compiled by Emerson C. Kelly, M.D., Department of Surgery, Albany Medical College. Vol. 1, No. 1. 78 pages. Price $10.50 a vol. (10 issues). Williams & Wilkins, Baltimore, 1936.

This publication is apparently devoted to two objects. One is to revive an interest in medical history, which is said to be most regrettably feeble—and we think this is quite truly so; the other is that it shall be of practical value in medicine. It is certain that there is no widespread interest in historical medicine, and we fail to see how the reprinting of even original papers can be expected to be regarded as of practical value in modern practice. But on the other hand it cannot be doubted that the few who are generally and continually interested in the history of medicine will look with great pleasure on this new venture. There have been other books on classical descriptions of disease, but this is much more ambitious. It will indeed be something to look forward monthly (for ten months in the year) to seeing the original papers of such men as Addison, Dupuytren, Graves, Murphy, Parkinson, to mention only a few.

This first number contains three papers by Sir James Paget admirably reproduced, with a short biography and a bibliography of his writings, which in itself is a striking commentary on his industry and breadth of interests.

BOOKS RECEIVED

Prevention and Treatment of Disease. William M. Stevens, M.D. 26 pages. Price 2s. H. K. Lewis, London, 1936.

Pathology. Eugene C. Piette, M.D., Pathologist and Director of Clinical Laboratories, West Suburban Hospital, Oak Park, Ill. Second edition, 263 pages, illustrated. Price $1.75. F. A. Davis Co., Philadelphia, 1936.

Modern Treatment and Formulary. Edward A. Mullen, P.D., M.D., F.A.C.S., Assistant Professor of Pharmacology and Physiology, Philadelphia College of Pharmacology and Physiology. 707 pages. Price $5.00. F. A. Davis Co., Philadelphia, 1936.

Allergic Diseases. Ray M. Balyeat, M.A., M.D., F.A.C.P., Associate Professor of Medicine, University of Oklahoma, and Ralph Bowen, B.A., M.D., F.A.A.P., Chief of Pædiatric Section, Balyeat Hay Fever and Asthma Clinic, Oklahoma City. Fourth edition, 516 pages, illustrated. Price $6.00. F. A. Davis Co., Philadelphia, 1936.

Applied Dietetics. Sanford Blum, A.B., M.S., M.D., Head of Department of Pædiatrics, San Francisco Polyclinic and Post-Graduate School. 408 pages. Price $4.75. F. A. Davis Co., Philadelphia, 1936.

A Health Education Workbook. Kathleen W. Wootten, M.A., Professor of Health, Head of Department of Health and Physical Education, Georgia State College for Women, Milledgeville, Ga. 273 pages. Price $1.50. A. S. Barnes, New York, 1936.

Manual of Pharmacology. By the late Walter E. Dixon. Eighth edition revised by W. A. M. Smart, M.B., B.S., B.Sc., M.R.C.S., L.R.C.P. 483 pages, illustrated. Price $5.50. Ed. Arnold, London; Macmillan, Toronto, 1936.

Food and the Principles of Dietetics. Robert Hutchison, M.D., LL.D., F.R.C.P., Consulting Physician to London Hospital, and V. H. Mottram, M.A., Professor of Physiology at King's College of Household and Social Science, University of London. Eighth edition, 634 pages, illustrated. Price $6.25. Ed. Arnold, London; Macmillan, Toronto, 1936.

A Dissertation on the Sensible and Irritable Parts of Animals. Albrecht von Haller. 49 pages. Price $1.00. Johns Hopkins Press, Baltimore, 1936.

Elementary Pathology. Keith S. Thompson, Pathologist, Selly Oak Hospital, Birmingham. 74 pages, illustrated. Price 10s. 6d. H. K. Lewis, London, 1936.

Keeping Your Child Normal. Bernard Sachs, M.D. 148 pages. Price $1.50. Paul B. Hoeber, New York, 1936.

Snow on Cholera. John Snow, M.D. 250 pages. Price $2.50. Commonwealth Fund, New York, 1936.

Applied Dietetics. Frances Stern, Chief of Food Clinic, Boston Dispensary. 263 pages. Price $3.50. Williams & Wilkins, Baltimore, 1936.

Statistical Methods for Research Workers. R. A. Fisher, Sc.D., F.R.S. Sixth edition, 339 pages. Price 15s. net. Oliver & Boyd, Edinburgh, 1936.